Profile of 150 Cases of Carcinoma Stomach in Tirunelveli Medical College Hospital

DISSERTATION SUBMITTED FOR

M.S. GENERAL SURGERY

DEGREE EXAMINIATION

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INTRODUCTION

Carcinoma stomach is one of the leading causes of morbidity and mortality accounting for 12% of deaths. There has been a decline in its incidence since 1930. Even though the incidence of distal gastric cancer is decreasing in western countries, incidence of proximal gastric cancers continue to increase.

The overall 5-year survival rates ranging from 53% in Japan to 10% in Eastern Europe.A better outcome is obtained in Japan due to an active screening programme resulting in earlier diagnosis and an aggressive surgical approach. The combination of chemoradiation therapy and curative resection increases disease free and overall survival duration which has led to recommend multimodality treatment in patient with systemic disease. Inspite of this multimodality treatment the morbidity and mortality continues to be high.

AIMS OF THE STUDY

This study was mainly to analyse cases of carcinoma stomach in our region which commonly present to the general surgeon in the department of general surgery , Tirunelveli medical college hospital, Tirunelveli.

The study was conducted in the following principles.

- To study the anatomical location of growth with respect to age and gender distribution
- To study the Etiology & Risk factors with respect to anatomical location.
- Study of H.Pylori association with carcinoma stomach.
- To study the sensitivity and specificity of various investigation modalities used.
- To study the Histopathological variety in relation to the site of growth
- To study the mortality and morbidity rate with reference to treatment modality.

MATERIALS AND METHODS

This study was conducted in the general surgery department , Tirunelveli medical college hospital ,Tirunelveli for a period of 30 months, from June 2006 to November 2008.

- 150 Cases of Carcinoma stomach were studied during this period.
- Details of 150 patients with Adenocarcinoma were retrieved and follow up of these patients was done with the surgical treatment modalities used and its outcome and survival.
- Cancer recurrence after surgery and cancers after GJ for peptic ulcer disease were excluded from this study.
- Age, gender, predisposing factors, clinical presentation and surgical treatment modalities used were recorded in a prestructured proforma.
- The site of the stomach tumours was recognized as involving one of the four sites. Namely

  1. OG Junction 2. Proximal Stomach (Cardia & Fundus)

  3. Body 4. Antrum

- Only those cases which received some form of surgical treatment were included in the study.

- Association of H.Pylori with gastric cancer study was carried out in eleven patients of histologically proved gastric carcinoma.

- The WHO classification of HPE report was combined with Lauren's HPE type in order to study the Histopalthology type in relation to tumour location.

DATA ANALYSIS

- The Commonest abdominal malignancy in our region is CA stomach.
- The Common site of involvement in CA stomach is Pyloric Antrum constituting 117 out of 150 cases.
- This was in concordence with the study of cherian et al (2007), Sivaraman et al.
- This study, is contradictory to the rising incidence of AdenoCA of proximal growth in the West, Iran, Sweden & Kerala

SITE DISTRIBUTION

Site	No of cases	%	1993, TVMCH
OG jn	6	4%	2%
cardia	7	4.6%	2%
Body	15	10%	24%
Antrum	117	78%	48%
Linitisplastica	5	3.3%	10%

- Proximal growth shows an increase of 2% when compared to the previous study.
- A decline in the incidence of body growth (10%) was found in this study.
- Antral growth continues to increase by 30% in this study.
- The study revealed that the distal growth is continue to be increasing our region than the proximal growth .

AGE DISTRIBUTION

Site	Age groups				
	<30	30-39	40-49	50-59	>60

OG jn			2	2	2
Cardia			2	3	2
Body		2	7	2	4
antrum	1	8	16	34	52
Linitisplastica			3		2

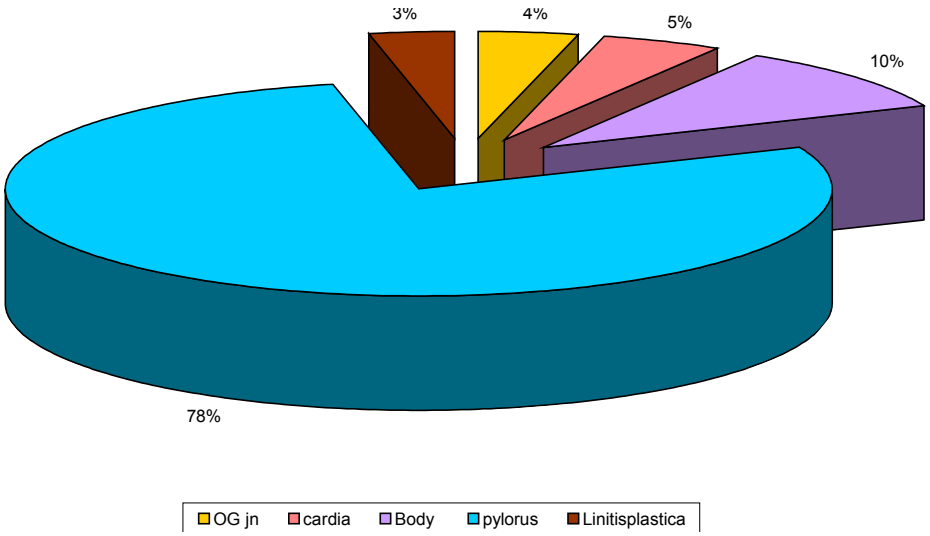
- The age group affected is evenly distributed in cancers involving OG jn above 40 years..

- Significant increase in the number of patients after the age of 50 years was seen in cancers involving pyloric antrum.

- Both the youngest age(24yrs) at presentation and oldest age(84yrs) of presentation were seen in Antral growth.

- This is concordance with the results of Lee et al and Kim et al.

SITE DISTRIBUTION



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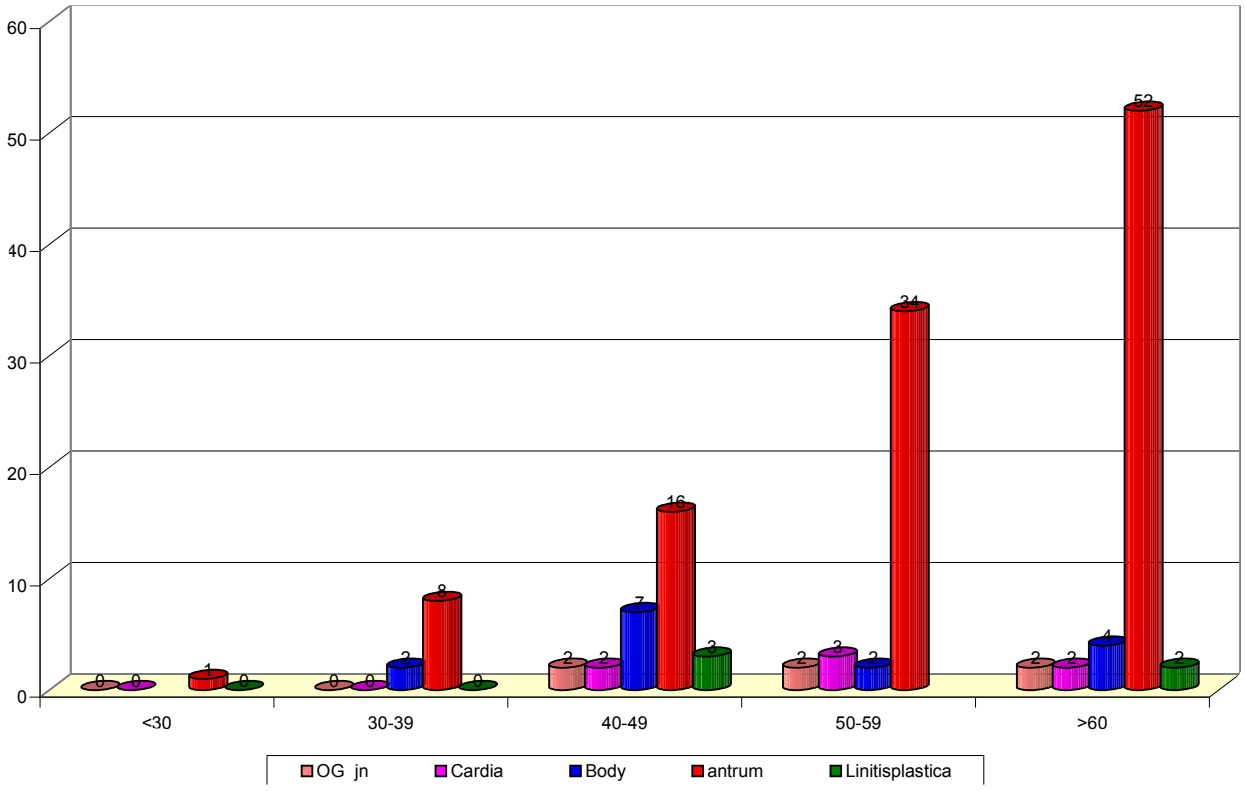


GENDER DISTRIBUTION

Site	Male	Female	Ratio
OG jn	3	3	1
Cardia and fundus	6	1	6
Body	11	4	1.2
Pyloric antrum	96	20	4.6
Linitis plastica	3	2	1.5

- The male : female ratio of cancers involving the proximal stomach, body, antrum and OG Jn was 6, 1.2, 4.6 and 1 respectively.
- This was not in concardance with the study of Cherian et al, where the ratio is 3, 2, 3 and 4 respectively.

MEAN AGE DISTRIBUTION

Site	Male	Female	SJE 2007 Male	SJE 2007 Female
OG jn	47.5	47	50.5	46.25
Cardia&fundus	51.4	45	55.47	50
Body	52.5	58.1	55.43	46.37
Antrum	56.7	60.1	55.5	52.86
Linitis plastica	51.3	57.5	-	-

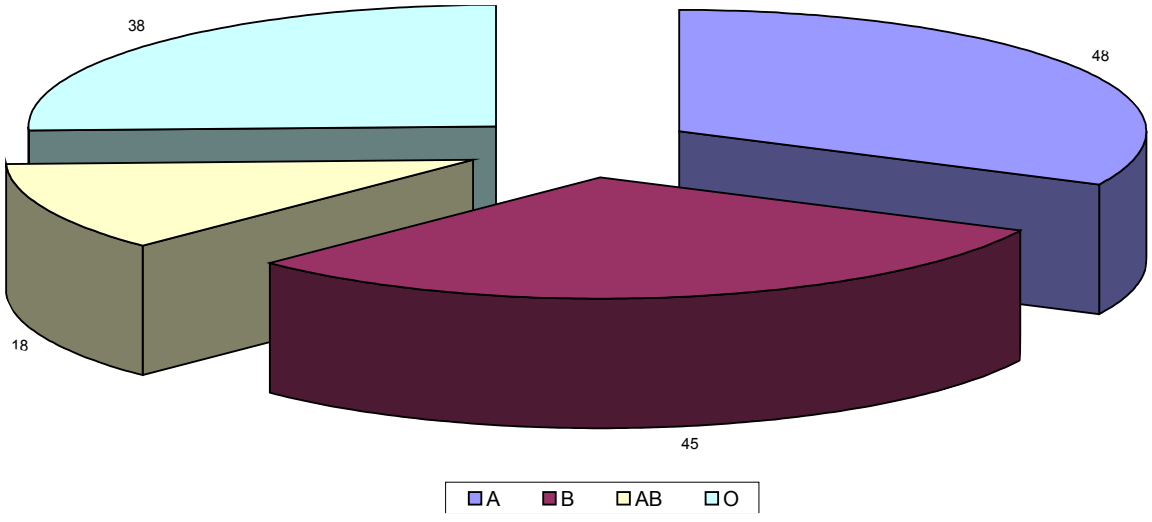
- Mean Age of occurrence is low in both sexes having proximal growths.
- This is in concardance with the study of Saudi gastroenterology 2007.

ETIOLOGICAL ASSOCIATION

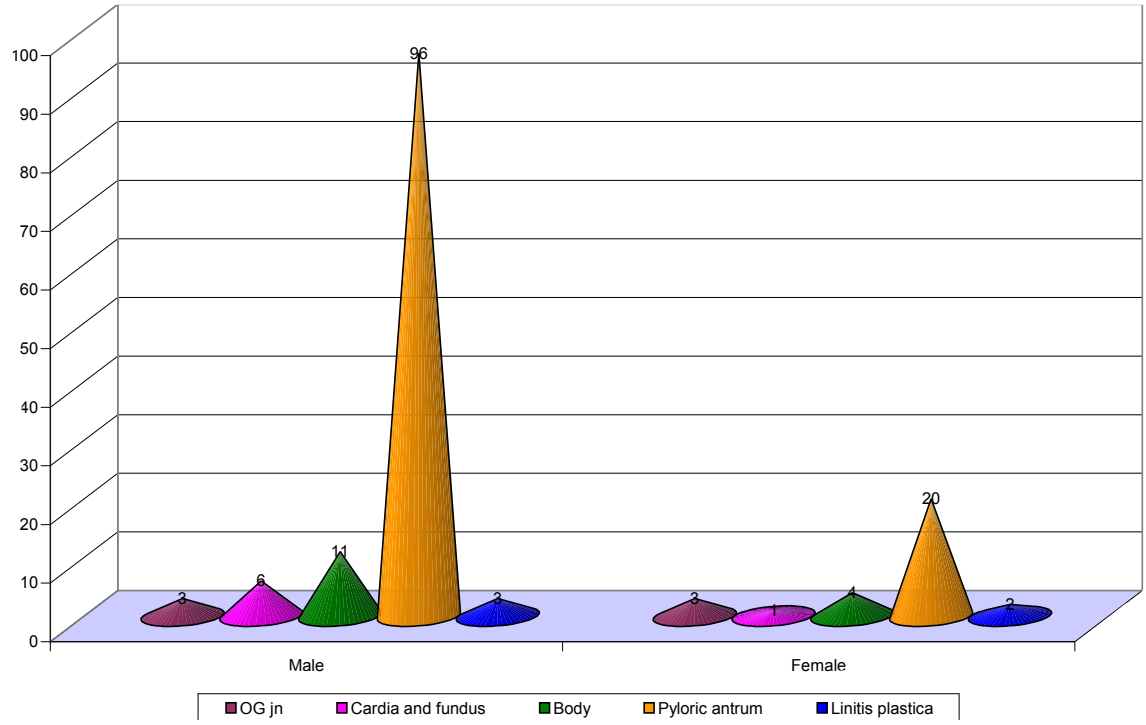
Site	Smoking	Alchohol	Smoked, spicy foods	Blood Grouping				Fatty food
				A	B	AB	O	
OG jn	3	1	2	2	3	0	1	6
Cardia&fundus	3	1	1	3	0	2	1	7
Body	3	2	12	2	3	4	6	5

Antrum	62	48	78	39	39	12	27	15
Linitis plastica	3	1	2	2	0	0	3	1

- Smoking contributes 100% association of cancers in OG Jn in males and 71.4% in cardia and 64.58% in Antral growth.
- Gammon et al, Inoue et al describes the association between smoking and proximal gastric cancers.
- Blood group A is the most common group affected by Carcinoma Stomach in our region. AIRD describes the association between ca stomach and Blood group A.
- Smoked and spicy food shows an increased association with antral growth.
- Fatty food has an increased association with proximal cancers.



GENDER DISTRIBUTION



BLOOD GROUPING

H.PYLORI STUDY

- 11 Patients underwent study of H.pylori by subjecting them to ELISA of IgG Antibody of H.Pylori kit

- About 5ml of blood was drawn from each patient,serum was separated and the IgG Antibodies were measured.

- The results were interpreted as positive if the value is above 20IU and negative if it is below 20IU.

- One patient with OG jn growth ,two patients with body of growth, eight patients with pyloric antral growth were subjected to study.

- The study includes seven patients of more than 60 years of age, two patients of fifty to sixty years of age, one patient below 50 years of age and one patient of thirty five years of age.

- Of these six patients are with blood group A, eight patients with intestinal type of lauren's classification, one patient with mucinous Adenocarcinoma and two patients with poorly differentiated Adenocarcinoma.

From this study ,the following results were obtained

- H. Pylori showed an association with intestinal type of 86.5%

- This is in concordance with study of Anderson et al of 90%

- Age group affected in H.pylori infection is above 60 yrs.

- This study corelates well with study of Feilding et al & Hunt et al, shows an association of age group affected above 60 yrs.

- H. Pylori shows an association with antral growth in 27.2% of patients and body of the stomach in 18% of patients

- Sibata et al shows an association of 68% with antral growth 50% with cardia growth and 66% with body of growth.

- H.Pylori shows an 50% Association with BLOOD Group A.

SENSITIVITY & SPECIFICITY OF INVESTIGATIONS

Site	Endoscopy	USG	BA meal	Ba swallow
OG jn	96%	0%	-	42.8%
Cardia&fundus	100%	0%	-	
Body	100%	20%	-	-
antrum	96.39%	90%	-	-
Linitis plastica	20%	40%	100%	-

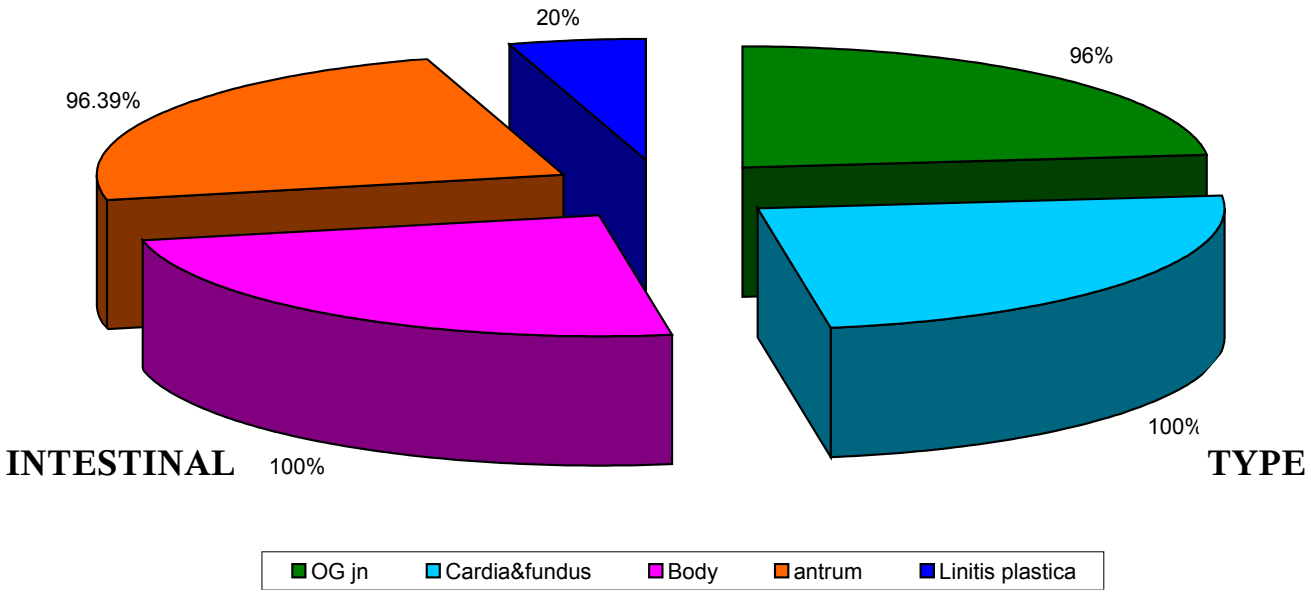
- Endoscopy was done for 140 cases
- Endoscopy and Biopsy shows 96.39% in detecting Antral growth and 100% in detecting Cardia & Body growth and 96% in detecting growth in OG Jn.
- USG has a sensitivity in detecting Antral growth as Antral wall thickening in 90% of cases.
- Ba meal has a sensitivity of 100% in detecting Linitis Plastica.

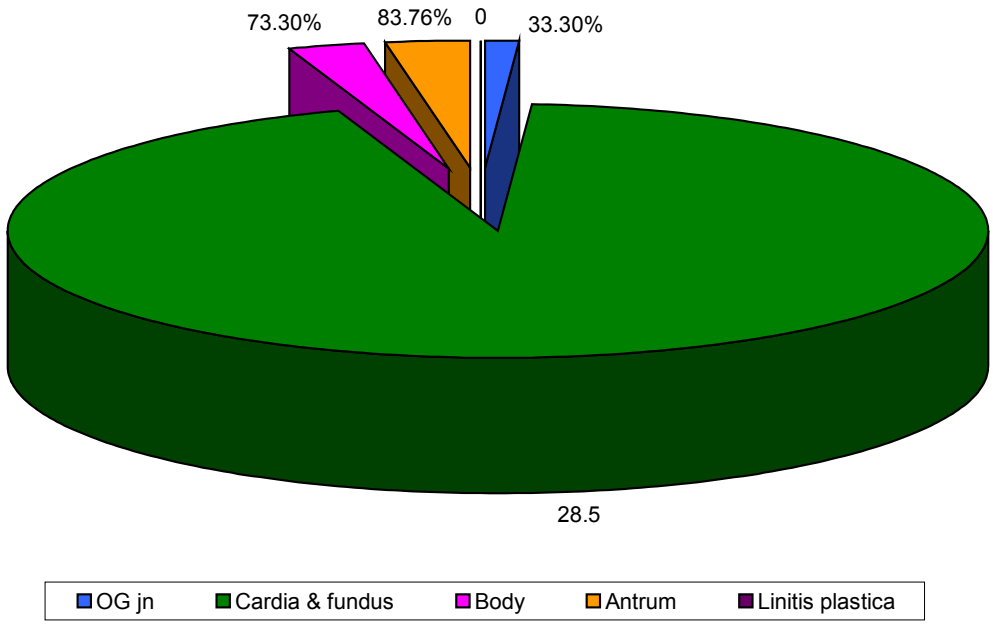
HISTOPATHOLOGY ASSOCIATION

Site	Intestinal type	Diffuse type	Mixed	Chandha, khan et al 2007		
				Intestinal type	Diffuse type	Mixed type
OG jn	2(33.3%)	4(66.6%)	-	-	-	-
Cardia & fundus	2(28.5)	3(66.6%)	2	5.3%	15.3%	-
Body	11(73.3%)	4(2.6%)	-	8%	15.3%	4%
Antrum	107 (91.45%)	7(5.9%)	3	28%	27.3%	8%
Linitis plastica	-	5(100%)	-	-	-	-

- The Intestinal type of Lauren's classification is found in 91.45% of cases of Antral Growth.
- The diffuse type contributes to 5.9% in growth of Antral cancers.
- The mixed type contributes 1% in Antral growth.
- The diffuse type is predominantly seen in proximal cancers and Linitis Plastica contributing 66.6%.

ENDOSCOPY AND BIOPSY




73.30%
83.76%
0
33.30%
28.5
OG jn
Cardia & fundus
Body
Antrum
Linitis plastica

Chandha et al and khan et al in 2007 describes intestinal type is more common in the pyloric antrum and cardia and fundus growth shows a predominance of diffuse type.

STAGING

Stage	No of patients	%
Stage 1a	-	-
Stage 1b	-	-
Stage 2	10	6.6%
Stage3a	11	7.3%
Stage3b	3	2%
Stage 4	126	84%

Site	Stage 1a	Stage 1b	Stage2	Stage3a	Stage3b	Stage4
OGjn			1			5(83.3%)
Cardia			1	1		5
Body			2	1	1	11
Antrum			6	9	2	100(66.6%)
Linitis						5(100%)

- The proximal tumours are usually advanced at presentation and have poorer long term prognosis -anderson et al

- In this study OG jn growth shows of 83.3% STAGE 4 disease &pyloric antrum shows 66.6% of STAGE 4 disease

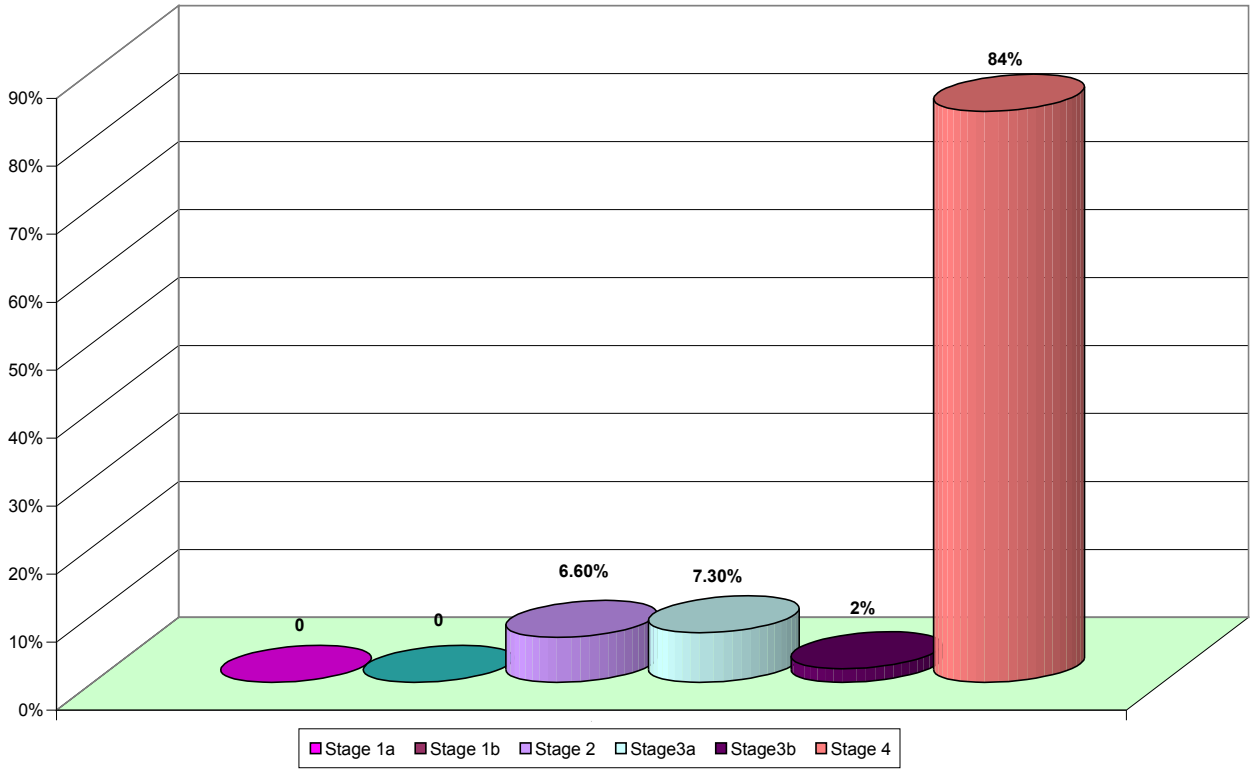
NODAL STAGING

Staging	No of patients	%
No	28	18.6%
N1	94	62.6%
N2	20	13.3%
N3	1	0.6%

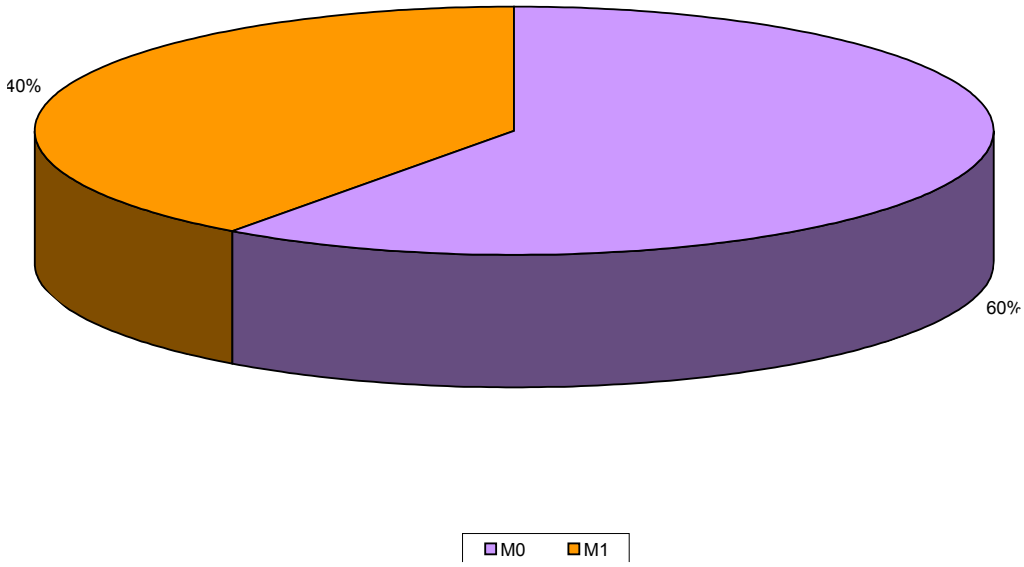
M STAGING

Staging	No of patients	%
M0	24	16%
M1	16	10.6%

STAGING



M STAGING



SURGICAL RESULTS

Surgery	No of cases	Morbidity	Mortality	Anderson et al Mortality
Total gastrectomy	8	12.5%	37.5%	8%
Sub total gastrectomy	8	-	-	10%
Distal gastrectomy	10	-		-
Distal gastrectomy and Roux en y	3	-	-	-
Palliative distal gastrectomy	44	-	2.8%	7%
Anteriorgastrojejunostomy	63	1.3%	9.5%	-
Feeding jejunostomy	14	-	2.8%	-

- Total gastrectomy was done for eight patients with a morbidity rate in two cases and mortality of three cases.
- The palliative procedure shows high morbidity may be due to disease process in my study.
- Anastomotic leak was found in six patients with 4%.
- .The most devastating complication is anastomatic leak in 3%-21% of cases - anderson et al
- Death due to co morbid illness in 4% of patients.
- Sub total gastrectomy and curative distal gastrectomy did not show any mortality in my study.

REVIEW OF LITERATURE

Carcinoma stomach is the one of the leading cause of cancer related death in world wide. It is the eighth most common cancer in US. It is the seventh most common cancer In India. There has been decline in its incidence since 1930. Although the incidence of distal gastric cancer is decreasing in the world, the incidence of proximal gastric tumors continues to increase.

The approximate incidence of gastric carcinoma in the United States is 10cases/1,00,000 people. In Japan -78 Cases/1,00,000 people. In India, cancer stomach is the 6th common cancer in male and 7th most common cancer in female.In TVMCH the incidence of ca stomach is 8.79% based on cancer registry during the period 2001-2002. In particular, gastric cardiac tumors are five times more common and non-cardia gastric tumors are twice as common in men as women. The incidence is also higher in low socio economic populations of developing countries. Survival rates in patients with gastric cancer is poor. The overall 5-year survival rates ranging from 53% in Japan to 10% in Eastern Europe. The analysis of the national cancer Data base of US revealed 6% to 12% better 5 year survival rate for women than for men, for patients with distal as opposed to proximal tumours. continues to be high.

HISTORY

Billroth, Wölfler	1881	First successful resection on human patient
Wölfler,nicolondi	1881	Performed antecolic gastrojejunostomy

Braun	1893	Described jejunojejunostomy as routine addition to gastrojejunostomy
Billroth,	1874	Billroth I - Distal gastric resection with anastomosis of Stomach and duodenum. Billroth II - Anastomosis of stomach with jejunum through transverse mesocolon

ANATOMY OF STOMACH.

For descriptive purposes ,the stomach is divided into

Lesser curvature

This continue with the right free border esophagus and forms the concave border of the stomach.

Greater curvature

This starts at the cardiac notch and forms the convex border of the stomach.

The fundus

This is the area above the horizontal line from the cardiac notch to the greater curvature of the stomach.

The incisura angularis

In a J shaped stomach this is the junction of vertical and horizontal parts of the lesser curvature

The body

This is the portion of stomach lying between the fundus and pyloric portion of stomach.

The pyloric portion

The distal one fifth of the stomach consists of pyloric antrum ,canal and sphincter .The pyloro duodenal junction is identified by the vein of mayo.

SURGICAL ANATOMY OF STOMACH

SKANDALAKIS ET AL divide the stomach into proximal gastric unit

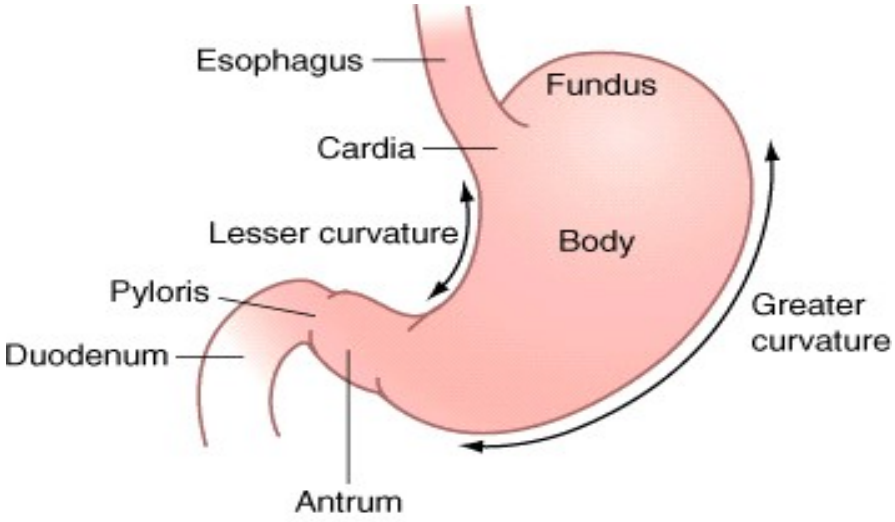
and distal gastric unit.

Proximal gastric unit consists of

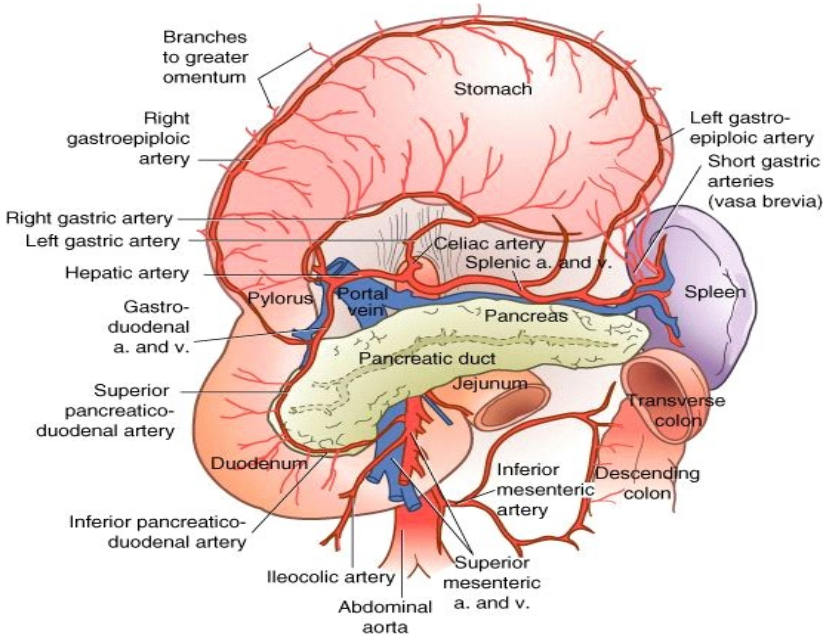
Distal esophagus - 0.5 -2.5cm

Og jn and esophageal hiatus

ANATOMY OF STOMACH



BLOOD SUPPLY OF STOMACH



Proximal stomach(cardia &fundus)

Distal gastric unit consists of

Gastric antrum

Pylorus

1st part of duodenum -first 2.5 cm of duodenum

Relations of proximal gastric unit

Lesser & greater curvature

The upper part of lesser sac

OG jn

Lesser curvature of body, GE jn and the abdominal esophagus are attached to the hepato gastric ligament and its contents .The greater curvature attaches to the upper part of greater omentum and several related splenic ligaments.like gastrosplenic and splenoleinal ligament.

Relations of distal gastric unit

The lesser curvature of antrum, pylorus and upper part of duodenum are attached to hepatogastric and hepato duodenal ligament

The greater curvature is attached to gastrocolic ligament.

Posterior relations of distal gastric unit

- Floor of lesser sac
- Transverse mesocolon
- head & neck of pancreas

- Aorta&celiac trunk & branches
- Celiac ganglion & plexus
- Hepatic triad
- Gastro duodenal artery

Anterior relations of distal gastric unit

- Anterior abdominal wall
- Medial segment of left lobe and anterior segment of right lobe of liver
- Transverse mesocolon
- Neck of gall bladder

BLOOD SUPPLY OF STOMACH

The four main arteries of stomach are

LEFT GASTRIC ARTERY

Arises from coeliac axis The left gastric artery commonly divides into an anterior and a posterior branch before attaining the lesser curvature[111] and, in such cases, the esophageal and cardioesophageal arteries may arise, variably, from either of those vessels; more commonly, however, the cardioesophageal artery arises from the anterior gastric branch.

After its cardioesophageal branch the left gastric artery usually curves downward to the

right and thereafter descends along the lesser curvature. As it descends, it bifurcates into an anterior branch which sends branches to the anterior gastric wall, and a posterior branch which, similarly, supplies the posterior gastric wall. A smaller continuation of the left gastric artery continued along the lesser curvature.

RIGHT GASTRIC ARTERY

The right gastric artery is a small branch which arises most commonly from the proper hepatic artery (50-68%), left hepatic artery (28.8-40.5%), common hepatic (3.2%), The right gastric artery gives origin to one or more suprapyloric branches. Anterior and posterior branches from these anastomose with infrapyloric vessels and with the supraduodenal artery, providing for the distal gastric unit (antrum, pyloric canal, first inch (2.54 cm) of the first part of the duodenum). The right gastric passes along the lesser curvature for about 4 to 6 cm, about 0.5 cm from the lesser curvature, before anastomosing with the left gastric. In about 13% of individuals, the right gastric artery provides origin for the supraduodenal artery.

RIGHT GASTRO EPIPLOIC ARTERY

The right gastroepiploic artery is a branch of the gastroduodenal artery (or its continuation) in most cases. It occasionally arises from the superior mesenteric artery or from the anterior superior pancreaticoduodenal artery. After giving origin to an infrapyloric branch, the artery passes along the greater curvature of the distal gastric surgical unit within the gastrocolic ligament. It gives origin to 8-18 singular or paired anterior and posterior branches for the gastric wall. The gastric branches of the right gastroepiploic artery pass mostly undivided in a submucosal position about one-fifth of the distance from the greater curvature.

They anastomose extensively with branches from the left gastric artery.[108] If the first infrapyloric branch is particularly large, the first following gastric branch tends to take its origin further around the greater curvature. In about 75% of individuals, the right gastroepiploic artery clearly anastomoses with the left gastroepiploic,[111] although not so profusely as is seen in the anastomoses with the left gastric artery.

The gastroduodenal artery ran in front of the common bile duct and descended along the posterior surface of the head of the pancreas

LEFT GASTRO EPIPLOIC ARTERY

The left gastroepiploic artery is the largest branch of the splenic artery.[56] It reaches the stomach at a point about halfway along the greater curvature, after it has already delivered several rather long branches to the more proximal part of the greater curvature. These branches reach the stomach by way of the gastrosplenic ligament.The left gastroepiploic artery gives off the left epiploic and the anterior epiploics. Together with similar branches of the right gastroepiploic, they form the arc of Barkow , which is reinforced also by the posterior epiploic branches of the inferior pancreatic artery .

VEINS OF THE STOMACH:

The veins of the stomach mainly accompany the arteries. Of particular importance is the left gastric or coronary vein,receives branches from the esophagus.

LYMPHATIC SUPPLY OF STOMACH

The lymph nodes concerned in the drainage of stomach are.

HEPATIC GROUP

Lymph nodes lie in the lesser omentum along the bile ducts and receive lymph from the liver and gall bladder.

SUB PYLORIC GROUP

Lie in the angle between first and 2nd parts of duodenum on the head of pancreas in relation to duodenal artery.Receive the lymph from right two thirds of greater curvature of stomach.

GASTRIC GROUP

Superior nodes - lie along left gastric artery and para cardial nodes

Inferior nodes - lie along the pyloric half of greater curvature of the stomach

PANCREATICOLIENAL GROUP

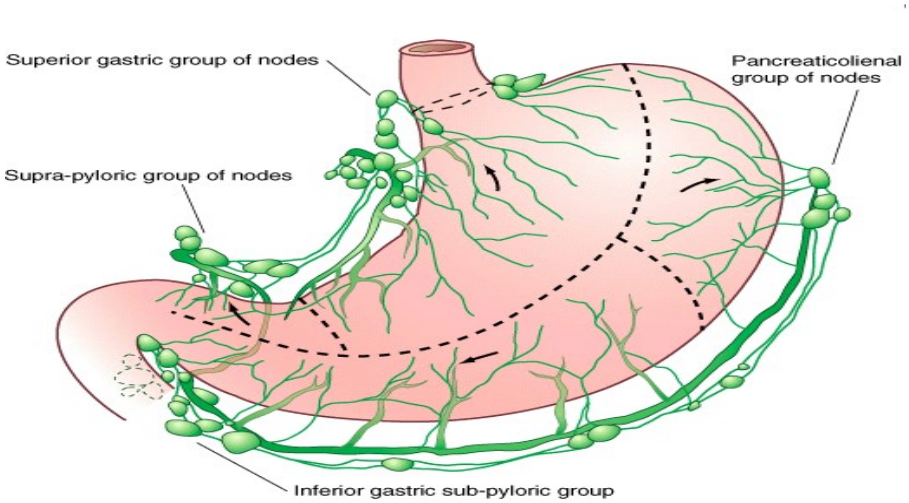
These lie along the splenic artery in relation to upper border of pancreas.

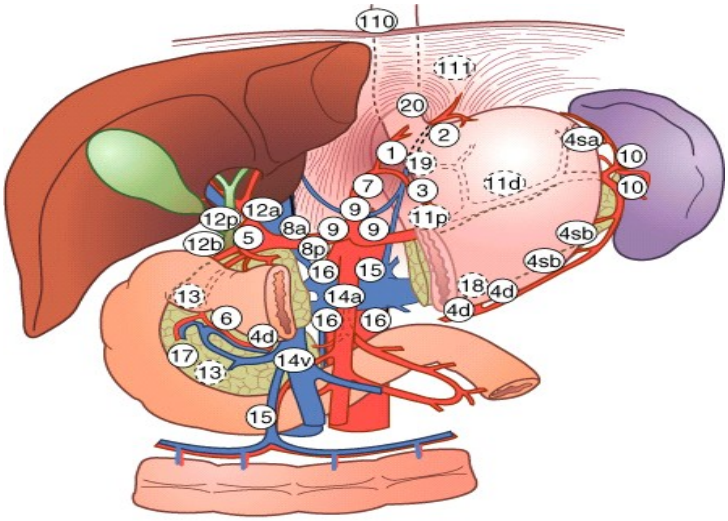
JAPANESE CLASSIFICATION OF LYMPH NODES

1. Rt. Paracardial

2. Lt Paracardial
3. Along Lesser Curve
4. Along Greater Curve
5. Suprapyloric
6. Subpyloric
7. Lt Gastric
8. Common HepaticCoeliac Axis
9. Splenic Hilum
10. Splenic Artery
11. Hepatoduodenal Ligament
12. Retropancreatic
13. Superior mesentric

LYMPHATIC SUPPLY OF STOMACH





JAPANESE CLASSIFICATION OF LYMPHATIC SUPPLY OF STOMACH

14. Middle colic
15. Para aortic
16. Anterior pancreatic
17. Inferior pancreatic
18. Infra diaphragmatic
19. Oesophagial Hiatus
20. Lower Paraoesophageal
21. Supra diaphragmatic

The gastric wall consists of the serosa, the muscular layer, the submucosal layer, and the mucosal layer.

SEROSAL LAYER

The serosa is nothing more than the peritoneum, a thin layer of loose connective tissue underlying a layer of simple squamous mesothelium.

MUSCULAR LAYER

The anatomist describes the three well-known layers of musculature: an outer longitudinal layer, middle circular layer, and inner oblique The outer longitudinal layer of muscle is present along the lesser and greater curvatures Its fibers are arranged into two groups. The first set is continuous with the outer smooth muscle layer of the esophagus. The fibers are best developed along the curvatures, ending proximal to the pyloric region. The second set begins in the body and passes to the right, with the fibers becoming thicker as they near the pylorus.

The middle circular layer rather uniformly encircles the entire stomach and "is the principal part of the muscular coat," according to Woodburne.[55] This layer becomes thicker at the pylorus, most distally forming the pyloric sphincter .

The inner oblique muscle layer, internal to the circular layer, is limited to the body of the stomach and is most developed near the cardiac orifice.

SUBMUCOSAL LAYER

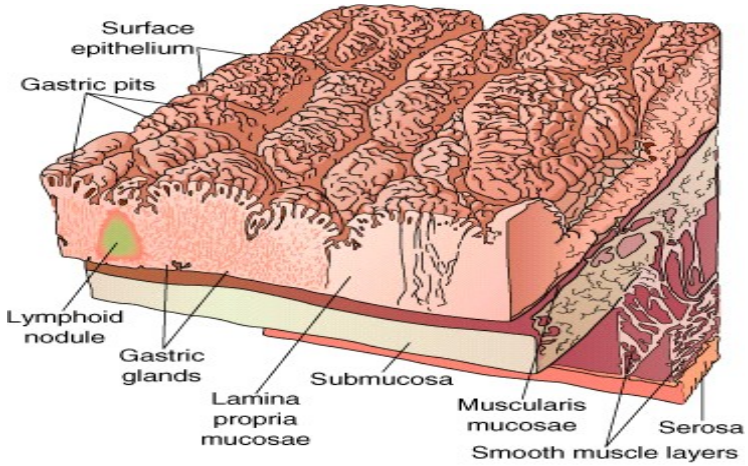
The submucosa is composed of loose, areolar connective tissue which connects the mucosa to the external musculature. It can be called the "vascular layer" (both arterial and

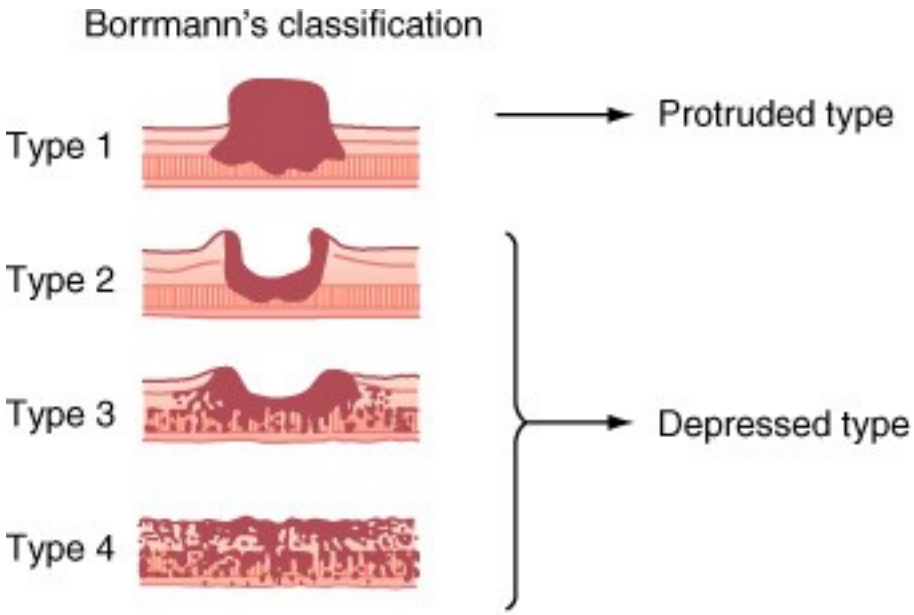
venous) of the gastric wall. It houses plexuses that have rich anastomoses through extensive ramifications that form excellent collateral flow. All the parts of the gastric mucosa receive blood from th ese submucosal plexuses except the lesser curvature.

MUCOSAL LAYER

- The distal esophagus is lined by stratified squamous epithelium, with mucous cells in the abdominal esophagus
- The cardia contains simple columnar cells

GASTRIC MUCOSA SURFACE





- The fundus and body contain two types of cells: parietal (oxyntic) acid-secreting cells and chief pepsin-secreting cells
- The lamina propria of the fundus and body is filled with branched, tubular gastric glands

RISK FACTOR

HELICOBACTER . PYLORI

Sibata et al & Huang et al found H.Pylori positivity is more associated with non-cardia gastric cancer. H.plyori was detected in 90% of intestinal type of cancer &32% of diffuse type cancer. It is also as associated with MALT and gastric Lymphoma. H.Pylori increases the risk of gastric cancer by impairing the bioavailability of vitamin C.

WARREN, in 1980 detected a curved s shaped bacilli in light microscopy. Initially it was recognized as campylobacter jejuni.in October 1989, the organisms was named helicobacter, because of the presence of sheathed flegelle,a unique fatty acid profile, different repiratory quionones.The isolation of H.Pylori from the gastric mucosa and the report of the organisms urease activity was postulated by MARSHALL.

H. Pylori is almost associated with inflammatory response.The spiral shaped organisms allows efficient motility in the mucus and in the gastric juice. The enzyme urease by breaking down urea in the gastric juice appears to generate enough bicarbonate and ammonium ions around the organisms to allow its safe passage through the gastric acid barrier to reach the mucous layer.Ammonia elevates the pH of the gastric mucosa from about 6-7.It is known to deplete aerobic cells of alpha keto glutarate, an essential substrate for the TCA acid cycle. Once attached to the mucus layer and the mucosa, H.Pylori, secretes soluble proteases and

phospolipase, which may be harmful to the mucus layer.

The vacuolating cytotoxin is a 87 kda protein , expressed in about 65% of H.Pylori strains and is responsible for creating vacuoles in the epithelial cells. The gene for cytotoxin protein is V ac A. The Vac gene is present in all H.Pylori bacteria but only produces an active protein in 65% of isolates.A second protein at 127 kda is called cytotoxin associated gene and is called Cag A gene. Cag A gene is a marker for vacuolating cytotoxin effect and is only present when Vac A cytotoxin is present. The organisms have been classified into Type I 0rganisms which have Cag A and Vac A which are more ulcerogenic and type II organisms that lack Cag A and do not produce cytotoxins. H. Pylori infection is associated with increase gastrin levels and decrease somatostatin levels, which has led to the development of atrophic gastritis ,a precancerous condition of gastric carcinoma. Epidemiological studies have demonstrate that there is an association of H.Pylori with gastric carcinoma . this infection has three fold to six fold higher risk for developing gastric cancer than those without infection.

The incidence of gastric carcinoma tends to be high in geographic regions were people consumes diets high in salt & smoked foods. The presence of nitrosamines in the smoked food is a carcinogen in developing the carcinoma.

Smoking has a five-fold increased risk of cardia and upper-third gastric cancer and a two-fold increased risk of distal gastric cancer compared to nonusers.

Male gender, Black race & low socio economic class are associated with Gastric

carcinoma.

Obesity is associated with Proximal gastric cancer.

Occupational hazard exists for metal workers is a associated factor for distal gastric cancer..

An associate between gastric carcinoma and blood group A was described in 1953 by…AIRD ET AL….

PREDISPOSING FACTORS

Gastric polyps particularly Villous Adenomas indicate increased risk of malignancy.

Pernicious Anemia is associated with 10% incidence of gastric cancer.

Mutations in CDH1…. Gene that encodes **E. Cadherin** is more common in diffuse type gastric cancer. Mutations in the CDH1 gene that encodes E-cadherin, an epithelial cell adhesion molecule, may be found in intestinal cancers but are more common in diffuse-type gastric cancers. Furthermore, defects in E-cadherin mediated cell adhesion are characteristic of diffuse-type gastric tumors. It also appears that a CDH1 gene mutation occurs in a cluster of patients with hereditary diffuse gastric cancer. Therefore, prophylactic gastrectomy is offered to carriers of these mutations. Virtually all carriers of this mutation so far have been found to harbor an early malignancy in the resected specimen, despite negative initial endoscopy findings, pointing to the advisability of the gastrectomy.

Vascular endothelial growth factor C (VEGF-C) is a glycoprotein that belongs to the VEGF family. VEGFs are cytokines that play an important role in angiogenesis. In gastric adenocarcinomas, expression of VEGF-C mRNA is associated with lymphatic invasion, lymph node metastasis, and possibly a less favorable outcome

P53 over expression revealed in more than 55% of tumors.

Previous partial gastrectomy & **Menetrier's disease** – Pre disposing factor for gastric cancer.

Hypogammaglobulinaemia is associated with gastric cancer.

PATHOLOGY

Approximately 10% of these tumours are polypoid fungating tumours, with a relatively good prognosis after aggressive surgical management. **Ulcerating or penetrating cancers,** which occur in more than 50% 0f patients, are sessile. **Superficial spreading carcinoma** is more unusual(6% of tumours). This tumour is diffusely infiltrative over a wide area,although it is predominantly confined to mucosa and submucosa. A further subgroup of diffusely infiltrative cancer is the LINITIS PLASTICA (leather bottle) carcinoma and carries particularly poor prognosis.

EARLY GASTRIC CANCER

The term early gastric cancer describes tumours confined to the gastric mucosa

and submucosa, irrespective of nodal status. Based on endoscopic appearance,(JAPANESE CLASSIFICATION),it is divided into three groups,type-1 protruding ,type -2 superficial ,and type-3 – excavated. Type -2 is divided into three subgroups,a) elevated b) flat and c)depressed Early gastric cancer accounts for 40% of all gastric cancers in Japan. In the west,early gastric cancer accounts for 9% of all cancers detected.50% of cases progress to advanced cancer within three years from the time of endoscopic diagnosis, and almost all patients develop advanced disease within five years.

CA STOMACH WITH ASCITES

METASTATIC CUTANEOUS NODULES IN CA STOMACH

ADVANCED GASTRIC CANCER

Advanced gastric cancer suggests invasion of the muscularis mucosa or beyond. It shows serosal involvement, invasion of adjacent organs,extensive nodal metastasis and secondary deposits usually within the peritoneal cavity.By, definition these tumours always exceed two cm in diameter (.BORRMANS CLASSIFICATION)

MICROSCOPIC APPEARANCE

The great majority of gastric cancers are Adenocarcinoma; other tumours are rare.

WHO CLASSIFICATION

The WHO classification based on morphology divides gastric cancers are into five types:adeno carcinoma,adenosquamous carcinoma, squamous cell carcinoma, undifferentiated carcinoma, and unclassified carcinoma. Adenocarcinomas contribute 95% of gastric cancers which arises from the mucous producing rather than acid producing cells of gastric mucosa The adeno carcinoma are divided into four patterns; papillary, tubular, mucinous, and signet ring, which may be divided by degrees of differentiation. In tumours presenting a mixed picture with varying degrees of differentiation in the same tumour ,is based on the predominant type.

Lymphomas , carcinoid, Leiomyosarcoma, GIST of stomach , adenosquanmous and squamous cell carcinoma comprise 5% of gastric cancers.

LAUREN'S CLASSIFICATION:

According to Lauren's classification, the two Histologic types of gastric carcinoma are intestinal and diffuse. The intertinal type have the tendency of Malignant cells to form glands. The diffuse type lacks organized gland formation, ie usually poorly differentiated and has many signet ring cells.The intestinal type carries the the more favorable prognosis and is more common in areas with a high incidence of the disease.

MING CLASSIFICATION

In this classification ,cancers were divided into an expanding type that produces nodules that compress adjacent tissue (Laurens intetinal type), and the infiltrative type that does not forms masses(Lauren's diffuse type)

CLINICAL PRESENTATION

Gastric adenocarcinoma is usually not associated with specific symptoms early in the course of the disease. Patients often ignore the vague epigastric discomfort and indigestion that portend the cancer and may be treated presumptively for benign disease for 6 to 12 months before diagnostic studies are performed. Rapid weight loss, anorexia, and vomiting are usually a sign of advanced disease. These presenting features are simply due to the presence of a partially obstructing lesion .

The epigastric pain is usually similar to the pain caused by benign ulcers and is often relieved by eating food; however, it can mimic angina. Dysphagia is usually associated with

tumors of the cardia or gastroesophageal junction. Antral tumors may cause symptoms of gastric outlet obstruction. Although very rare, large tumors that directly invade the transverse colon may present with colonic obstruction. Physical examination will reveal a palpable mass in up to 30% of patients.

Signs of Metastasis disease is found in 10% of patients.

The most common indications of distant metastasis are a palpable supraclavicular lymph node (Virchow node), a mass palpable on rectal examination (Blumer shelf), a palpable periumbilical mass (Sister Mary Joseph node), ascites, jaundice, or a liver mass. The most common site of hematogenous spread is the liver; tumor also frequently spreads directly to the lining of the peritoneal cavity

Gastric tumors may be associated with chronic blood loss, but massive upper gastrointestinal bleeding is rare. In Japan, the high incidence of gastric cancer has led to routine endoscopic screening; as a result, in that country, more than 50% of gastric cancers are diagnosed at an early stage.

STAGING SYSTEM

The Pathologic staging system currently in use worldwide is the American joint committee on cancer TNM staging system with the addition of the term R status to denote residual disease remaining after resection

TNM STAGING

The American joint committee on cancer (AJCC) has designated staging by TNM

classification.

PRIMARY TUMOUR

- **Tx :** Primary tumour cannot be assessed
- **T0**: no evidence of primary tumour
- Tis: carcinoma in situ: intra epithelial tumour without invasion of the lamina propria
- T1:Tumour invades lamina propria or submucosa
- T2:Tumour invades the muscularis propria or the submucosa
    1. T2a:Tumour invades muscularis propria
    2. T2b:Tumour invades subserosa
- T3: Tumour penetrates the serosa(visceral peritoneum) without invading adjacent structures
- T4: Tumour invades adjacent structures.

REGIONAL LYMPH NODES

- Nx: Regional lymph nodes cannot be assessed
- N0: No regional lymph node metastasis
- N1:Metastasis in 1-6 regional lymph nodes
- N2:Metastasis in 7-15 regional lymph nodes
- N3:Metastasis in more than 15 regional lymph nodes.

DISTANT METASTASIS

- Mx:Distant metastasis cannot be assessed
- M0:No distant metastasis
- M1:Distant metastasis.

AJCC STAGING

Stage 0 (Tis,N0,M0)

Stage 1a (T1, NO,MO)

Stage1b (T1,NI,MO;T2A,N0,M0;T2B,NO,MO)

StageI I (T 1,N2,MO;T2A,NI,MO;T2B,N1,M;T3,NO,MO)

StageIIIA (T2A,N2,MO;T2B,N2,MO;T3,NI,MO;T4,NO,MO)

StageIIIB (T3,N2,MO)

StageIV (T4,N1,MO; T4,N2,M0; T4,N3,MO; T1,N3,MO;T2,N3,M0; T3,N3,M0; Any T,ANY N,M1)

RESIDUAL DISEASE

R status described by Hermanek and wittekind in 1994 is commonly used to describe the tumour status in a patient following resection and is designated following pathologic evaluation of resection margins.

R_0 - No residual gross disease and Negative microscopic margins.

R_1 - Microscopic residual disease only

R_2 - Gross residual disease.

INVESTIGATIONS

UPPER GI ENDOSCOPY:

Differentiation between benign & malignant gastric ulcers at endoscopy can be difficult and several biopsies are taken (ideally six) from all parts of ulcer. Diagnostic accuracy approaches 100% if 10 samples are taken.

BARIUM MEAL

It gives a better impression of anatomy of tumour, and the degree of obstruction. It is also helpful for diagnosis of linitis plastica which may be missed at gastroscopy.

CT SCAN

Abdominal and pelvic CT is performed in the overall staging of patients. CT chest may be required for complete staging of proximal gastric tumours. The major limitations of CT as a staging tool are in the evaluation of early gastric tumors and small metastases in the Liver. The overall accuracy in determining tumors stage is 66% to 77%. CT can be used to determine nodal stage in 25% to 86% of patients.

ENDO ULTRASOUND

The overall staging accuracy of EUS is 75%. It correctly identifies T2 leisons (38.5%) of the time. It is better at identifying T1(80%) and T3(90%) lesions. The accuracy of EUS in the nodal evaluation is 65%.

LAPAROSCOPY

The Clinical benefit of laparoscopic staging after CT staging is that it identifies CT occult Metastatic diseases in 23% to 37% of patients.

PERITONEAL CYTOLOGY

Cytologic analysis of peritoneal fluid obtained by peritoneal lavage may identify occult carcinomatosis. Patients with positive peritoneal cytology findings have a prognosis similar to that of patients with macroscopic visceral (or) peritoneal disease(3 – 9 month median survival)

LYMPHATIC MAPPING:

Gives the essential role of nodal status in gastric cancer staging. Mapping agents such as radiocolloid with (or) without vital dye and activated carbon particles have been used. The identification rate varies from 90% from 100%.

POSITRON EMISSION TOMOGRAPHY:

Positron emission tomography, estimate tumour metabolism on the basis of the uptake of radiotracer – flurodeoxyglucose – used a staging tool for gastic cancer. This technique may reveal CT-occult metastases and may be used to assess response to Neoadjuvant therapy.

TUMOUR MARKER

CA 72-4 is highly specific to gastric cancer and may be more reliable as a tumour marker than CEA for gastric cancer. Elevated level of CA 72.4 is more frequent than CEA in stage III, IV patient and with Peritoneal metastasis.

H. PYLORI TESTS.

Tests	Sensitivity	Specificity
Histology (Geimsa stain)	93-99%	95-99%
Culture	72-92%	100%
Urease test	89-98% ;	93-98%
Urea breath test	95-98%	95 -98%
Serology ELISA	88-89%	86-95%
PCR	95%	95%
Stool antigen test	95-99%	90-95

SURGICAL TREATMENT

EARLY GASTRIC CANCER

Endoscopic Submucosal Resection

For mucosal cancers, approximately 3% will be expected to have positive lymph nodes and selectively justify the use of an endoscopic submucosal resection (ESMR). Patients with submucosal cancer, where the risk of nodal positivity can reach 20%, would be better considered for limited laparoscopic resections or limited open operations. Lymph node metastasis in both these situations is associated with larger tumor size, undifferentiated histology, and the presence of histologic lymphatic or blood vessel invasion. As a working rule lymph node metastases are rare if the tumor is less than 2 cm in diameter and well differentiated regardless of the depth of mucosal invasion.

Laparoscopic Resection

Laparoscopic resection is being used increasingly for early-stage cancer. This is

performed essentially by an extragastric approach after appropriate tattooing of the lesion endoscopically so as to ensure the ability to identify the lesion and the adequacy of the resection. The procedure can be prolonged, and it is essential that clear margins be obtained. Progressively more advanced procedures such as distal gastrectomy are being performed with the laparoscopic hand-assisted approach using a minilaparotomy

Proximal tumours

This tumours accounts for about 50% of all gastric carcinomas. These tumours are usually advanced at presentation and associated with poorer long term prognosis.

Siewert classification of OG junction tumours

Type I **:** Associated with Barrett oesophagus (or) true esophaged cancer growing into the OG junction

Type II **:** True junctional tumours that lie within 2cm of squamo columnar junction.

Type III **:** Cancer within subcardial region of stomach.

Type I **:** Treatment option for this tumour is oesophageal resection with 10 cm margin clearance.

Type II **:** oesophago gastrectomy

Type III **:** Total gastrectomy with reconstruction

There is no evidence that an extended gastric resection above and beyond complete clearance of the primary tumor improves survival; i.e., total gastrectomy does not improve survival over distal or proximal gastrectomy, provided that the tumor is removed completely with negative transection margins (R_0 resection). The extent of the gastrectomy therefore is site-

dependent and focuses on complete removal of the gastric carcinoma with preferably a 4- to 5-cm margin from the gross edge of the tumor. Clearly, anatomic limitations influence this margin because in antral lesions close to or involving the pylorus, only a limited portion of the duodenum can be removed. In similar fashion, a lesser extent of uninvolved esophageal margin may be acceptable provided complete histologic resection is possible.

Midbody Tumours

These account for 15% to 30% of all gastric cancers.

Treatement option for this tumours also total gastrectomy with regional lymphadenectomy.

Distal Tumour

These tumours account for approximately 35% of all gastric cancer. The standared operation for these lesions is distal subtotal gastrectomy with appropriate lymphadenectomy. Subtotal gastrectomy entails resection of approximately three fourths of the stomach, including the majority of lesser cuvature.

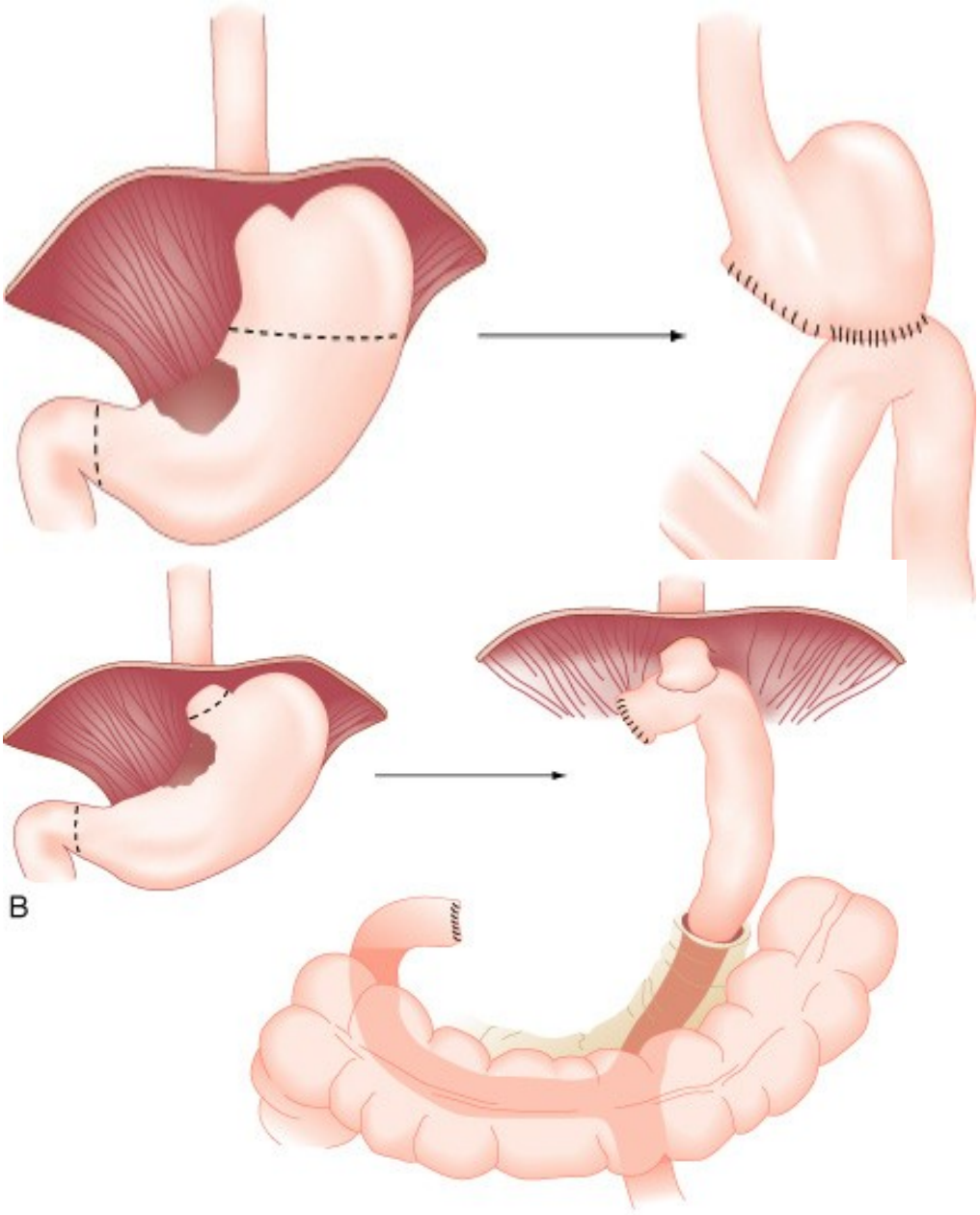
Extended organ resection is reserved for patients with apparently node-negative T4 lesions, in which complete resection requires resection of the invaded portions of the diaphragm, pancreas, spleen, adrenal gland.

SURGERY

TOTAL GASTRECTOMY:

(If the tumour involves the stomach diffusely or arises in the body of the stomach and extends to within 6 cm of the cardia or distal antrum) Removal of stomach with greater & leeser omentum along with nodes en bloc with Roux en y reconstruction. In a total gastrectomy, early mobilization of the left lateral segment of the liver will be necessary to evaluate the extent of a more proximal lesion .However, once the right side of the esophagus is isolated, then theesophagus can be encircled, and the left paracardial nodes and the paraesophageal nodes can be reflected inferiorly with the specimen In this situation, the short gastric vessels need to be divided. The left crus is skeletonized, and the left adrenal gland is identified commonly ,transect the stomach with the placement of a proximal Satinsky vascular clamp, which allows ready stabilization of the esophagus In proximal lesions, a frozen section of the esophageal margin is performed. In locally invasive extensive proximal lesions, the mucosal margin can be benign, but extraesophageal extension can be found in the periserosal tissues. Once this specimen is removed, the jejunal loop is isolated and divided with the GIA stapler .The small intestine for the Roux-en-Y anastomosis is divided with the stapler. Attention should be paid to ensure adequate vascularity of the limb, and some require careful transillumination to be sure that the arterial arcade is maintained without subsequent tension. The reconstruction can be performed by a direct end of

SUB TOTAL GASTRECTOMY WITH BILLROTH – II - ANASTAMOSIS



ROUX EN Y OESOPHAGO JEJUNOSTOMY

esophagus to jejunum anastomosis It is helpful in the sutured anastomosis to place a single stabilizing suture in the posterior aspect of the esophagus, suturing the posterior aspect to the

posterior surface of the jejunum, which stabilizes and aligns the appropriate. An alternative reconstruction uses the EEA stapler .

PROXIMAL GASTRECTOMY

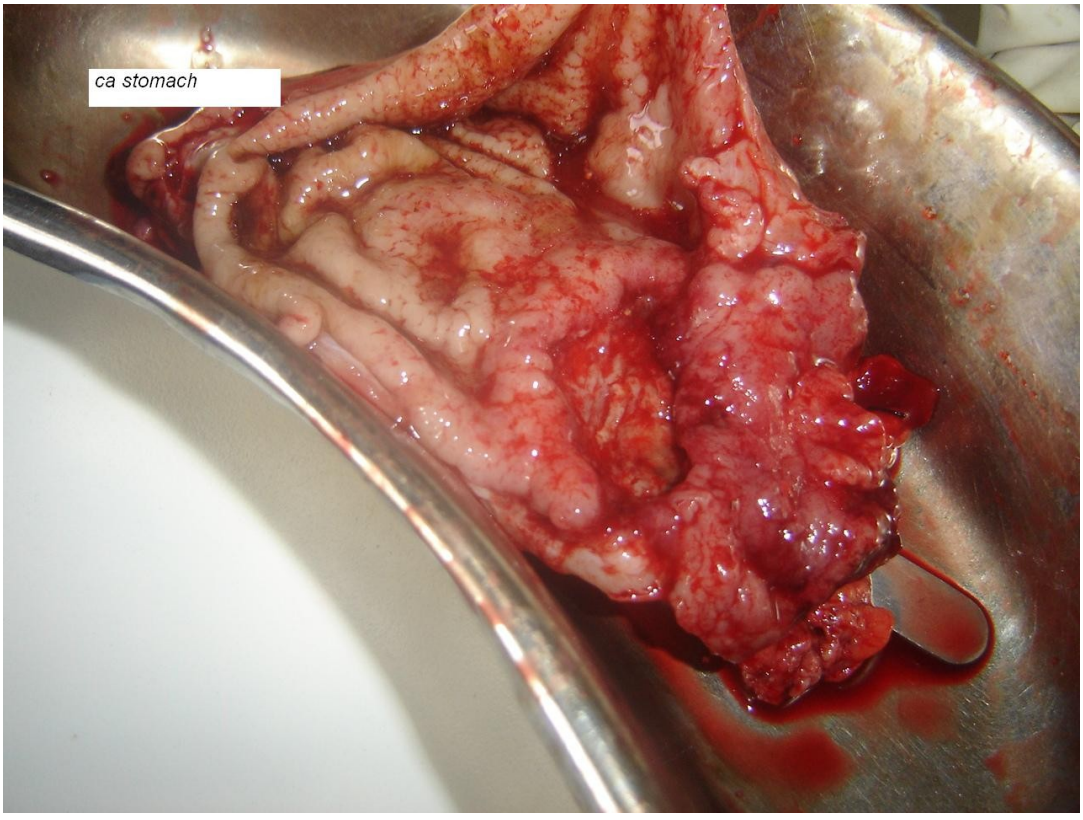
An simple midline incision is made in the abdomen

The left lateral segment of the liver is taken down and retracted, the esophageal hiatus is identified, and the soft tissue around the diaphragmatic hiatus is incised. The extent of the lesion is appreciated rapidly, and on occasion, local invasion into the crura of the diaphragm may need crural resection. The retroperitoneal attachments to the diaphragm on the left side then are incised, coming down toward the anterior surface of the left adrenal gland. Once the esophagus is mobilized, it can be retracted upward and forward, and a decision can be made as to the extent of resection required.

For cardiac lesions that are locally extensive, the spleen is mobilized and taken with the specimen. In more localized lesions, the short gastric vessels are taken close to the splenic hilum to enable greater mobilization. On the right side, the right crus is cleared, and all tissue is taken back to the celiac axis origin. The section then is carried forward along the anterior surface of the celiac axis to identify the left gastric artery. In the majority of

PARTIAL GASTRECTOMY

CUT SPECIMEN OF CA STOMACH



patients, all lymph nodes along the lesser curvature can be dissected readily and reflected back toward the proximal gastrectomy with the specimen. Care must be taken not to devascularize the lesser curvature. An appropriate distal site on the greater curvature is identified, and the stomach is divided with two passages of the GIA-60 or TA stapler. This can be done in an oblique fashion so as to create a relative tube of the distal stomach .

SUB TOTAL GASTRECTOMY

(If the proximal luminal resection margin is 5- 6 cm) .Removal of 80% of distal stomach leaving the short gastric vessels intact to ensure splenic preservation.Reconstruction is by either roux en y or billroth II or polya.

DISTAL GASTRECTOMY:

(Antral growth with 5cm marginal clearance) .Removal of less than 80% of stomach with reconstruction.

PALLIATIVE PROCEDURES

(For advanced gastric cancer to minimize bleeding and gastric out let obstruction)

- Gastrectomy
- Anterior gastro jejunostomy
- Laser or argon beam tumour ablation with laser recanalization.
- Endoscopic placement of coated metallic stents.

COMPLICATIONS OF SUGERY:

The most devastating complication is anastomatic leak, which occurs in 3% to 21% of

patients. Dumping syndrome will develop in 10% of patients.early dumping typically occurs 15 to30 minutes after meal and includes diaphoresis,abdominal cramps, palpitations and watery diarrohea. Late dumping is usually associated with hypoglycemia and hyperinsulinemia. The management include dietery habits and if refractory, a somatostatin analog.

Other complications are

- Bleeding – 0.3-5%
- Pulmonary 3-55%
- Infection 3-22%
- Anastomotic leak 3-21%
- Cardiac 1-10%
- Renal 1-8%
- Pul. Embolus -1-4%

OUT COME OF SURGERY

The overall 5 yr survival rate is 10-21 percent who undergo potentially curative resection has a slightly better prognosis with a 5 year survival rate of 24% to 57%. The 5 year survival rate who under go curative resection is 50% .The overall locoregional recurrence rate was 38% the median time to death from the time of recurrence was 6 months.

Neoadjuvant therapy

The advantages of Neoadjuvant chemotherapy is decreased tumour seedling at surgery and tumour sensitivity to a chemotherapeutic regimen.drugs used are etoposide, cis platin, 5-fluro uracil . The clinical response rate of combination of etoposide ,cisplatin and 5Fu range

from 21% - 31%.

Adjuvant chemo therapy

The adjuvant chemotherapy of 5Fu was associated with a survival benefit of 95%.

Post op External beam Radiation therapy

The combination of 5Fu with 40gy external beam radiation therapy versus RT alone showed improved survival. The three year survival rate for chemo RT were 52% compared to surgery only group 40%

Management of Advanced Disease

About 20-30% patients present with stage IV disease. The 5yr survival rate for patients with stage IV disease is zero. Because of this low cure rate and advanced disease at presentation, palliation is an essential component of gastric cancer Management.

Palliative surgery

Surgery achieves good palliation less than 50%of the tome. The procedure include palliative Distal gastrectomy with acceptable mortality rates. Palliative total gastrectomy & esophago gastrectomy should be approached with greater caution because the morbidity from these procedures is higher 4%.

Palliative chemotherapy

The Combination regimen included FAMTX(5Fu, doxorubicin, Methotrexate) FEMTX (5Fu, epirubucin, Methotrexate) and ELF(etoposide, leucovorin, &5Fu) patients who received combination chemotherapy had better survival.

Pallative Radiation therapy:

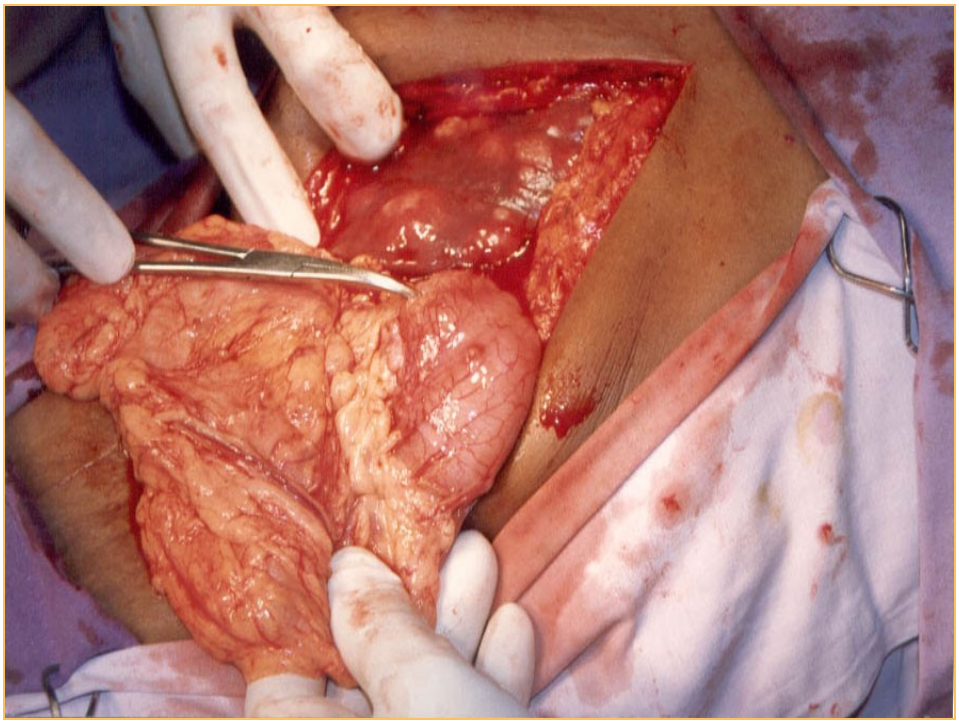
No large prospective trial has demonstrated a long term benefit from radiation therapy.

Palliation by Endoscopic means include laser recanalisation (or) simple dilation with (or) without stent placement can be used to treat obstruction.

Intraperitoneal Hyperthermic Perfusion

This procedure is a combination of hyperthermia & Mitomycin C. Fujimoto et al reviewed that patients treated with perfusion survived longer(1 year survival rate 80% vs 34%).

CA STOMACH WITH LIVER SECONDARIES



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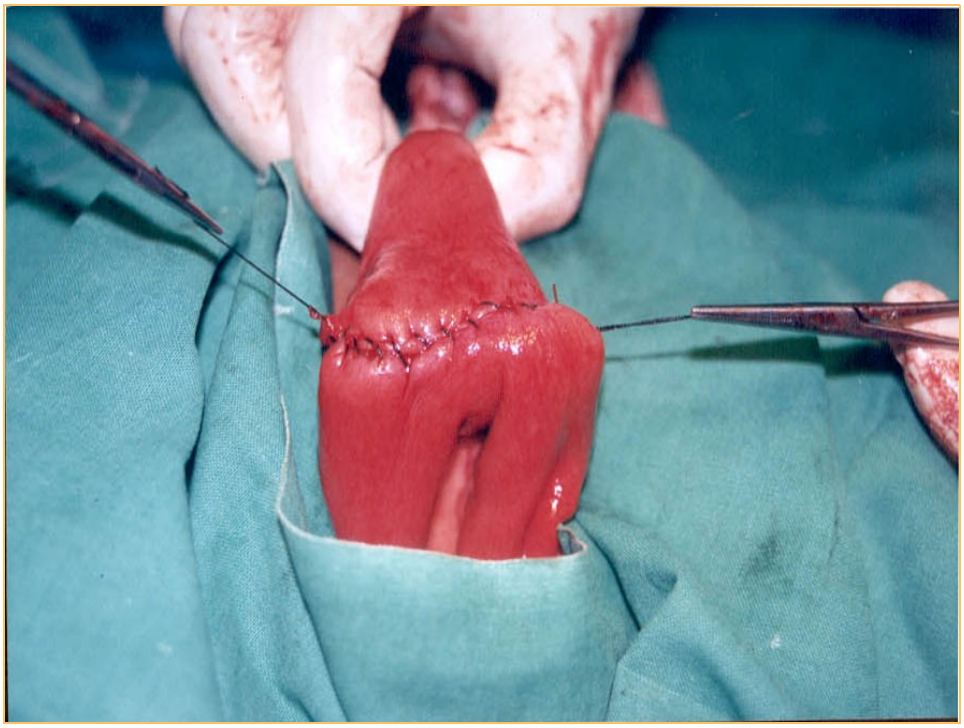

Immunotherapy

Machara et al reported a combination of chemotherapy and intraperitoneal injections of OK-432 had a long survival rate. Other immunotherapy's are mycobacterium derived polysaccharides.

Gastric Lymphoma

The Stomach is the most common site of lymphoma in GIT. The average age of patients is 60 years.the most frequent site ofpresentation are pain, weight loss,bleeding and fatigue. H.Pylori appears to be a causative agent is the development of gastric lymphoma and MALT

lymphoma. Endoscopy detect the lesion is 80% of cases as sub mucosal infiltration.Other Lab test include lactate dehydrogenases and B2 Micro globulin determination. The pathological type of NHC is B cell Type & diffuse histiocytic type. Treatment include Acid Suppression therapy, Metrogyl and Antibiotic.Anti H. PYLORI treatment is necessary for eradication of H. p ylori. External beam irradiation and chemo therapy is offered to those who do not respond to an antiobiotic eradication and high risk patients. Gastric resection is rarely performed for pt. with MALT lymphomas.surgery is necessary in some cases to confirm diagnosis.Surgical resection is curative in patients with localized disease, although half of resected patients require chemotherapy.If surgery is performed,an attempt is made to resect the area grossly involved with lymphoma but leave grossly normal stomach intact. Negative margins are not necessary for cure. Surgery alone produces survival rate of 82%, where as radiation therapy produces five year survival rate of only 50%. 10- year survival rates of 80% have been seen in patients with Ann Arbor staging of stage IE and Stage II .Patient with lymphoma are initially treated with chemo therapy of CHOPP regimen. Radiation and surgery are reserved for patients who do not have a complete response to chemotherapy.

Gastric Carcinoid

Gastric carcinoid constitute only 2% of carcinoid tumors. gastric carcinoids are classified into three types . Type I carcinoids are associated with type A chronic atrophic gastritis, Type II carcinoids are associated with Zollinger-Ellison syndromewith multiple endocrine neoplasia syndrome-I, and type III are sporadic gastric carcinoids. Type I carcinoids are the most common type of gastric carcinoid and are found predominantly in

women. These patients have typically have elevated plasma gastrin levels and low gastric acid production. Type II gastric carcinoids are the least common type, accounting for 8% of cases with an equal distribution. Even they are associated with Zollingar Ellison syndrome in conjunction with MEN- I they share features with type -1 carcinoids,such as location in the fundus, multricentricity, elevated plasma gastrin level and small size.Type III carcinoids , which are sporadic carcinoids, represent 23% of cases and are found predominantly in men; The mean age of patients at diagnosis is 49 years. Patient may complain of histamine producing symptoms, such as cutaneous flushing,bronchospasm,itching and lacrimation. The sporadic tumours are single, solitary ,large and located in the antrum or fundus of stomach. The natural course of type III carcinoids is more aggressive with hepatic metastasis found at diagnosis in upto 50% of cases.

Gastric carcinoids are diagnosed by both biochemical and histologic means. Upper gastrointestinal endoscopy, including endo ultrasound, is also essential to evaluate the number, size ,extent and location of lesions. Extensive gastric biopsy should be performed in those with suspected gastric carcinoids, checking for histologic chromogranin, features of dysplasia,mucosal atrophy,and the degree of mucosal invasion.it appears that the rate of diagnosis correlates with the number of biopsys performed.CT of the abdomen is essential to exclude metastasis to the liver and nodal involvement.

The type of gastric carcinoid dictates the nature of treatment .for patients with either type I or type II carcinoids and with tumours less than 1 cm or with fewer than three to five lesions, treatment typically consists of an endoscopic polypectomy or endoscopic mucosal

resction.Antrectomy or local excision may be performed in a young patient with an elevated gastrin level who has recurrent or multifocal disease.. This treatment will decrese gastrin levels and frequently leads to the regression of the tumours. Older patients with many lesions may be followed if the lesions are small. For diffuse or recurrent disease, a completion gastrectomy may be recommended, depending on the patients age and course of the disease.Type III disease should be considered for en bloc resection with lympadenectomy, depending on the tumour size.It appears that five year survival rate for patients with type I and type II gastric carcinoids are between 60% and 75%.in contrast, the five year survival in patiets with type III gastric carcinoids is less than 50%. Follow up in patients with gastric carcinoids primarily consists of plasma chromogranin estimation. Plasma gastrin and urinary 5-HIAA levels may also be evaluated, although urinary 5-HIAA levels.

GASTRO INTESTINAL STROMAL TUMOURS

GISTs are the most common mesenchymal tumors of the stomach, with the stomach being the most common location of these tumors in the gastrointestinal tract (40%), followed by the small intestine (32%). GISTs of the stomach are predominantly found in males, with a median age at diagnosis of 63 years. The presenting features in these patients are varied and include bleeding (38%), abdominal pain (11.8%), and rupture (1%); 12% of patients have no symptoms. The median size of GISTs of the stomach is 6 cm.

Regardless of the location of these tumors in the stomach, R0 resection remains the treatment of choice, conferring a 5-year survival of 55%. Several studies have shown that the features recommended in the NIH guidelines, tumor size (>5 cm) and mitotic index (>5/50 hpf), are associated with a less favorable outcome. Tumor location in the gastroesophageal junction or fundus, ulceration, coagulative necrosis, and mucosal invasion were found to be associated with a poor outcome .Antral tumors, however, were found to be associated with a more favorable outcome .Those patients with unresectable or metastatic disease are offered treatment with Imatinib (Gleevec, an oral tyrosine kinase inhibitor). They may then be re-evaluated for potential resection if they demonstrate response to treatment.

OBSERVATION

- Cancer stomach is one of the commonest abdominal malignancy in our region.

- The incidence of distal growth continues to be increasing in our region.
- The incidence of CA stomach in distal part usually present with age above 50yrs.
- Cigarrete smoking showed increase in proximal cancers when compared distal cancers.
- The association between Blood group A and Carcinoma Stomach correlate with the study of AIRD ET AL.
- Endoscopy and Biopsy have a high sensitivity in picking up all subsite growths.
- The intestinal type and in association with distal growth have a favourable prognosis in operable cases.
- Stage 4 disease is common in proximal cancers which shows poorer prognosis in over all survival
- Gastrectomy with its acceptable morbidity is associated with good quality of life, whenever the tumour is operable
- Total Gastrectomy carries a high morbidity and mortality when compared to subtotal and partial Gastrectomy

CONCLUSION

- The causes of Distal stomach cancer continues to be commoner than proximal cancers among Indians.

- The Etiology of carcinoma stomach tends to be different etiology in both proximal and distal cancers.

- H.Pylori has a 45 .5% association of ca stomach and it is mostly associated with distal growths.

- Endoscopy has a high sensitivity in detecting all subsite growths but least sensitivity in detecting early cancers.

- Most cases present with stage 4 disease which shows absence of screening of this disease.

- Curative subtotal and distal gastrectomy is associated with good quality of life whenever the tumour is operable.

- Mortality is due to increase in duration of surgery and associated co-marbid illness and anastomotic leak.

MASTER CHART

S. No	Name	Rel	IPNo.	Age/ Sex	SE	SM	AL	Diet	Subsite	Pain	Vom/ DYSP HAGIA	Mass	L/N	L/A
1	Asokan	H	17640	65/M	800	√	√	M	Antrum	√	√			√
2	Pattanaasari	H	47730	60/M	800			M	Antrum	√	√		√	
3	Gurusamy	H	30628	50/M	800			M	Antrum	√	√			
4	Rajammal	H	28355	50/F	800			M	Antrum	√	√			√
5	Kalliammal	H	33605	55/F	800			M	Antrum	√	√	√		√
6	Pillaikani	H	21600	54/M	800	√		M	Antrum					
7	Mayillappan	H	14828	64/M	800	√		M	Antrum		√	√		√
8	Ayyadurai	H	2733	45/M	800	√	√	M	Antrum	√	√			√
9	Palaniappan	H	281395	62/M	800	√	√	M	Antrum	√	√		√	√
10	Muppidathi	H	35131	55/M	800			M	Antrum	√	√			
11	Muhtukutti	H	19729	49/M	800			M	Body(LC)		√	√	√	√
12	Murugan	H	26428	55/M	800	√	√	M	Antrum	√	√	√	√	√
13	Babu	H	36204	67/M	800	√		M	Antrum	√	√			√
14	Muthiahdevar	H	35208	65/M	800	√		M	Body(GC)			√		

15	Thiraviampillai	H	5705	70/M	800			M	Antrum	√	√	√		√
16	Ravikumar	H	37841	24/M	800			M	Antrum	√	√			
17	Mohammed	m	36686	47/m	800	√	√	fat	antrum	√				√

S. No	dehydration	MassPalpable	FullnessVague	VGP	Liver	GB	Ascites	Pelvic Deposits	BL GP&TY	USG/CT	Endoscopy	Biopsy	Bameal	H PYLORI	
1.	√								A+ve	Antral wall thickening	Antral Growth Goo	Adeno ca			Antral LN+ Fixed
2.				√					AB+ve	Antral wall thickening	Antral Growth Goo	Adeno ca			Antral G 2° A
3.				√					O+ve	(N)	Antral Growth	Adenoca			Antral Mes dep
4.		√ (E)		√					B+Ve	(N)	(N)	-			Growt
5.		√ (RHC)		√					AB+ve	Antralwall thickening 2° Liver Ascites	Antral Growth	Adeno ca			Grow 2° A
6.	√	√ (E)		√					AB+ve	Antralwall thickening	Antral Growth	Adeno ca			Antral L
7.	√			√					B +ve	Antralwall thickening para ao rtic LN	Antral Growth				Antral LN fixed t TC, PA,
8.	√								B+ve	Antral wall thickening	Antral growth	Adeno carcinoma			Antral LN infi par
9.	√			√					B+ve	Dilated stomach	Pre pyloric ulcer	Adeno carcinoma			Antral Peri
10.				√					B+ve	Duodenal thickening	Antral Growth & Goo	Adeno carcinoma			Antral C
11				√					B +ve	Antral wall thickening	GOO	Adeno carcinoma			Grou Pancreas
12		RHC		√	3cm				B +ve	Antral wall thickening, PALN,2°Liver	Antral Growth & Goo				Grou Pancreas TC,LN,
13		E		√					O+VE	Antral wall thickening	Antral Growth				Antral Mes dep
14		E					√		A +ve	Growth Body Ascites 2° liver	Growth Body	Adeno carcinoma		+	Grow Ascites
15		E		√					O +ve	Antral wall thickening,PALN,	Pyloric Antrum	Adeno carcinoma			Growth A to pancr Mes dep
16.	√	LHC							A +ve	N	Antral Growth	Poorly differentiated Adeno carcinoma			Growth A to pancr
17															

18	Natarajan	H	39830	58/M	800	√	√	M	CARDIA	√	Dysph agia			√
19	Samuvel	C	35767	50/M	800	√	√	M	Antrum	√	√			
20	Mariappan	H	14578	64/M	800	√	√	M	Antrum		√			
21	Muthiah	H	23988	57/M	800	√	√	M	Antrum	√	√			√
22	Shanmugavel	H	27093	61/M	800	√		M	Antrum	√	√	√		
23	Muthiahdevar	H	26818	61/M	800	√	√	M	Antrum	√				
24	Soundarrajan	H	27778	40/M	800			M	Antrum	√	√			
25	Balaiah	H	21981	55/M	800	√		M	Antrum	√	√			√
26	Vellammal	H	3988	55/F	800			M	Antrum	√	√	√		√
27	Velu	H	15037	68/M	800			M	Antrum	√	√		√	√
28	Ramakrishnan	H	51154	60/M	800			M	Antrum	√	√			√
29	Kanagaraj	H	18898	45/M	800	√	√	M	Antrum	√	√		√	√
30	Velkani	C	18974	52/F	800			M	Body(GC) LINITIS		√	√	√	√
31	Seenidurai	H	20148	50/M	800	√	√	M	Body LINITIS		√	√	√	√
32	Muthupandi	H	24804	65/M	800	√		M	Antrum	√	√			√
33	Lakshmi	H	30615	50/F	800			M	Antrum		√		√	√
34	Narayanan	H	21064	60/M	800			M	Antrum	√	√			√
35	Ganapathi	H	14009	50/M	800	√		M	Antrum	√	√			√
36	Alphonse	C	13893	50/M	800			M	Antrum	√	√			√

18	√			√					O+ve	Mass body of stomach	Body LC				Growth A to pancr Mes dep
19	√			√					O+ve	Ectopic Kidney GB Calculus	Growth Antrum				Growth A to panc
20				√					B +ve	Distended Stomach	Growth Antrum				Growt
21	√			√					O+ve	Antral wall thickening	Growth Antrum				Growth A to pancr
22	√	E					√		O +Ve	Antral Growth PALN	Growth Antrum	Poorly differentiated Adeno carcinoma			Growth A to pan
23		E		√					O+ve	Antral wall thickening 2°Liver	Growth Antrum	Adeno carcinoma			Gr Antrur LN,A
24				√					A+ve	N	Antral Growth				Growt mes LN,
25		E			4cm		√		O+ve	Antral wall thickening 2°Liver Ascites	Antral Growth GOO				Growth As
26	√	E		√					A+ve	Antral wall thickening	Antral Growth GOO			+	Growth A to
27	√		E	√					B+ve	Duodenal Pathology	Antral Growth				Growth A As fi
28			E	√					B+ve	Antral Wall thickening	Antral growth				Growt LN A
29		LHC					√		AB+ve	Antral growth LN ascites +	Anthral growth	Poorlydiffer Adeno carcinoma			Gr Antrur L
30	√	LHC					√		AB+ve	ascites	Anthral Growth, Linnitus, plastica	Poorlydiffer Adeno carcinoma	√		Contracte Stoma Ova
31	√	E(RHC)					√		A +ve	Mass epigastrium	Linitis Plastica	Adeno ca	√		Antra Body t
32	√	E			4 cm				B+ve	Antral Wall thickening	Antral Growth	Mucinous Adeno ca			Antral Py
33		E							AB+ve	Antral Wall Thickening,2° liver	Antral Growth				2° Antral Gr
34	√		E	√					AB+ve	Antral Wall LN, Ascites +	Antral Growth				Antra to Bod pancre
35	√		E	√					A +ve	Ascites GOO MRD	Antral Growth	Adeno ca			Growt As
36				√					O+ve	Antral Wall Thickening	Antral Growth				Antra

37	Shamugathai	H	37868	58/F	800			M	Antrum	√	√	√		
38	Sudalai	H	39760	74/M	800	√	√	M	Antrum	√	√			√
39	Arunachalam	H	3095	60/M	800			M	Antrum	√	√	√		
40	Vedhamanikam	H	49220	55/M	800			M	Antrum	√	√			√
41	Thirumaki	H	16078	63/M	800	√	√	M	Antrum	√	√			
42	Ramalakshmi	H	22240	47/M	800			M	Antrum	√	√		√	√
43	Dharmar	H	36087	58/M	800	√	√	M	Antrum	√	√		√	√
44	Thirumalai	H	16078	63/M	800	√	√	M	Antrum	√	√		√	√
45	Ramaih	H	27873	40/M	800	√		M	Antrum	√	√			
46	Grace	C	18080	45/F	800			M	Antrum	√	√			
47	Andidevar	H	31637	60/M	800	√		M	Antrum	√	√	√		
48	Narasiman	H	9276	55/M	800	√		M	Antrum		√	√		√
49	Nagalingam	H	25936	54/M	800	√		M	Antrum		√	√	√	√
50	Rawathy	H	28113	50/F	800			M	Antrum				√	√
51	Ramachandran	H	52332	40/M	800			M	Antrum	√	√			√
52	Muthammal	H	33603	57/M	800			M	Antrum	√	√			√
53	Ayyanu	H	50078	65/M	800	√	√	M	Antrum	√	√	√		√
54	Msooth	M	31106	60/M	800	√		M	Body(GC)	√	√	√		
55	Subbiah	H	36270	75/M	800	√	√	M	Antrum	√				

37	√	LHC							O+ve	2° liver	Antral Growth	Mod. diff Adeno ca			Antral 2° liver par
38	√			√					O+ve	Antral Wall Thickening,Ascites	Antral Growth				Antral Fixed As
39		E		√					O+ve	Antral Wall Thickening	Antral Growth Goo	Adeno ca			Antral Fixed
40				√					O+ve	Antral Wall Thickening	Antral Growth	Adeno ca			Antral
41	√		E	√					B+ve	Antral Wall Thickening	Antral Growth				Antral Perige
42	√		E	√					B+ve	Antral Wall Thickening	Antral Growth				Antral C
43		E							O+ve	Antral Wall Thickening					Antral C
44		E		√					A+ve	Antral Wall Thickening	Antral Growth				Antral Gro mes
45				√					B+ve	N	Antral Growth	Adeno ca			Antral
46				√					B+ve	N	Antral Growth	Adeno ca			Antral C
47		ERHC		√					A+ve	Antral Thickening ascites +	Antral	Adeno ca			Antral C As
48		E			4 cm				AB+ve	Antral Thickening Liver Ascites	Antrum	Poorly Diff Adeno ca			Antral C Liver
49		E			3 cm				B+ve	Antral Thickening	Antrum				Antral C Liver L
50	√	E		√	3cm				AB+ve	Antral Thickening 2° Liver	Antrum	Adeno ca			Antral C 2°Live Par
51			√E						O+ve	Antral Thickening	Antral Growth				Antral Gr
52		√E					√		AB+ve	2° Liver ascites	Antrum Body				Antral asc panc
53				√			√		B+ve	Antral Thickening	Antral Growth				Antrur
54	√	E	E						AB+ve	Antrum Thickening 2°liver	N				Antrum E
55	√								O+ve	Ascites R+Pl Effu……..	N				Antrur

56	Muthah ayyar	H	31878	67/M	800	√		V	Antrum	√	√	√		
57	Durairaj	H	25389	56/M	800	√	√	M	Antrum	√		√		√
58	Muthiah	H	218324	84/M	800			M	Antrum	√	√	√	√	√
59	Sudalai	H	14342	64/M	800	√		M	Antrum	√	√	√		√
60	Sankaranaryan	H	38225	35/M	800	√	√	M	Antrum	√	√		√	√
61	Yesuvadian	C	3550	65/M	800	√	√	M	Antrum	√	√		√	√
62	Ramasubhu	H	20315	55/M	800	√	√	M	Antrum	√	√			
63	Subramanian	H	19426	70/M	800	√	√	M	Antrum	√		√		√
64	Muthusamy	H	91484	45/M	800	√	√	M	Body(Gc)	√				√
65	Murugan	H	20326	50/M	800	√	√	M	Cardia	√	Dysphagia	√	√	√
66	Narayanan	H	19252	45/M	800	√	√	M	ANTRUM	√	√			
67	Abraham	C	21382	54/M	800	√	√	M	AntrUM	√	√			√
68	Mahalingam	H	24529	38/M	800	√	√	M	Antrum	√	√			
69	Jeyankani	H	14206	48/F	800			M	Body(LC)	√	√	√		
70	Rangitham	H	22236	46/F	800			M	Body(GC)	√				√
71	Paramasivan	H	20313	60/M	800	√		M	Antrum	√	√			√
72	Ramasubbu	H	4298	43/M	800	√		M	OG jn	√	Dysphagia			
73	Ganapathy	H	40239	72/M	800			M	Body(GC)	√				√
74	Arumugam	H	30803	58/M	800	√		M	Antrum	√	√			√

56	√	E			2 cm				O+ve	Antrum Thickening 2˚ liver ascites	Antral Growth				Antrum liver p
57		E			2cm		√		O+ve	Antrum Thickening 2˚liver	Antral Growth				Antrum l
58	√	E			4cm		√		B+ve	Antral 2˚liver LN	Antral Growth				Antrum As
59		E			3cm				AB+ve	Antrum 2˚Liver LN	Antral Growth				Antrun LN A
60							√		B+ve	Antralwall thickening	Antral Growth				An
61	√		E				√		B+ve	Antralwall thickening	Antral Growth	Adeno ca			Antra LN+ Fixe
62			E				√		O+ve	Antralwall thickening	Antral Growth	Adeno ca			Antral G mesente
63	√	E	E				√		B+ve	Antralwall thickening ln +	Antral Growth				Antr As
64			LHC						AB+Ve	(N)	Growth Body GC	FNAC LN Adeno ca			Grow
65	√	E							A+ve	(N)	Fundal growth				Growt
66	√	E					√		B+ve	Antral growth	Antral Growth	Adeno ca			Antrum
67	√			√					O+ve	Antral thickening	Antral Growth	Adeno ca			Antral L
68			√						AB+ve	Normal	Body LC	Adeno ca			B
69			E						A +ve	Growth	Body LC	Adeno ca			Bo
70									O+ve	Antral wall thickening	Body LC				Bo
71			E	√					A+ve	Antral wall thickening	Antral Growth	Adeno ca		_	Antrum
72		E							AB+ve	N	Cardiac Growth	Adeno ca			Ca
73			E						A+ve	Antral wall thickening	Body GC	Adeno ca		+	Body G
74		E							A+ve	Antral wall thickening	Antrum	Adeno ca			Antral Gr Pancreas

75	Murugan	H	25549	49/M	800	√		M	Body(GC)	√				
76	Lakshmi	H	99969	47/F	800			M	Antrum	√	√			√
77	Ganapathy	H	32797	62/M	800			M	Antrum	√	√			√
78	Subbiah	H	23465	65/M	800	√	√	M	Antrum	√	√	√		√
79	Shanmugam	C	26217	60/F	800			M	Linitis plastica	√	√			
80	Kandiahdevar	H	49520	60/M	800	√		M	Antrum	√	√			
81	Mani	H	17314	60/M	800	√		M	Antrum	√	√	√		
82	Ganapathy	H	31909	66/M	800	√		M	Antrum	√	√			√
83	Narasingamal	H	21870	30/F	800			M	Antrum	√	√			
84	Mani	H	17414	60/M	800	√	√	M	Antrum	√	√	√	√	√
85	Sappani	H	49413	43/M	800	√		M	Body(GC)	√				
86	Subramani	H	17575	55/M	800	√	√	M	Antrum		√		√	√
87	Mariammal	H	39539	67/F	800			M	Body(GC)	√	√		√	√
88	Ganapathy	H	60604	67/M	800	√	√	M	Antrum	√	√	√		√
89	Manivelraj	H	5434	40/M	800	√	√	M	Antrum	√	√	√	√	√
90	Valliammal	H	57286	65/F	800	√	√	M	OG JN	√	Dysphagia			√
91	Thriumalai	H	34679	67/M	800	√	√	M	Antrum		√			√
92	Arjunan	H	37623	50/M	800	√		M	Antrum	√	√	√		
93	Thayammal	H	33860	55/F	800			M	CARDIA		Dysphagia	√		

75			E						O+ve	Antral wall thickening	Body GC	Adeno ca			B
76			√						B+ve	Antral wall thickening	Antrum				An
77		E							O+Ve	Antral wall thickening	Antrum	Poorly differentiated Adeno ca			An
78		E			2 cm				A+ve	Antral wall thickening, 2˚ Liver PALN	Antral Growth				Antrum
79		E		√					O+Ve	Antral wall thickening, 2˚ Liver		Adeno ca			2˚ Live
80		E		√					A+ve	Antral wall thickening, 2˚ Liver	Antral Growth	Adeno ca			Antrum 2
81		√					√		O+ve	Antral Thickening ascites	Antral Growth				Antrum
82	√		E						B+ve	Antral wall thickening	Antral Growth	Adeno ca Well differentiated			Antr
83				√					B+ve	Antral wall thickening	Antral Growth				Antr
84	√	E		√	4cm		√		A+ve	Antral wall thickening	Antral Growth				Antrum
85			E						O+ve	N	CT Body GC				Body
86		E					√		A+ve	N	GOO	Adeno ca Welldifferentiated			Antrum PE Rou
87	√			√					O+ve	Antral wall thickening	Antral Growth	Adeno ca			B
88	√	E			2cm				O+ve	Antral wall thickening,2˚ liver	Antral Growth				
89		E			3cm		√		B+ve	Antral wall thickening	Antral Growth				
90		E		√			√		AB+ve	N	Body GC				Growth B +PD +P pan
91	√	√		√			√		O+ve	Free Fluid LN Antral Growth	Antral Growth	Adeno ca			Liver 2° A TC in
92	√	√		√			√		B+ve	Free Fluid LN Antral wall thickening	Antral Growth	Adeno ca			Growt Ascitic f De
93		√			3cm				B+ve	Hepatomegaly IHBR dilatation PH nodes	Growth Body of stomach	Adeno ca			Growt stomach no

94	Arumugam	H	26655	52/M	800	√	√	M	Antrum	√	√			√
95	Thangaraja	H	34538	60/M	800	√	√	M	Antrum	√	√			
96	Muthusamy	H	41489	45/M	800	√		M	Body(GC)	√				√
97	Petchikumar	H	49496	73/M	800	√	√	M	Antrum	√		√		
98	Venkatachalam	H	49485	52/M	800	√		M	OG JN		Dysphagia	√		
99	Ganesan	H	7903	64/M	800	√	√	M	ANTRUM	√				
100	Pandi	H	5161	45/M	800			M	Body(GC)	√				
101	Selvaraj	H	6158	57/M	800	√		M	Growth Cardia		Dysphagia			
102	Syedmohamed	M	31863	60/M	800	√		M	Pyloric growth	√	√	√		
103	Paramasivan	H	29576	65/M	800			M	Antrum	√	√	√		√
104	Joseph	C	36392	65/M	800	√		M	Antrum Growth	√	√	√		
105	Vairamuthu	H	62501	60/M	800	√	√	M	Antrum	√	√	√		
106	Ganesan	H	7903	64/M	800	√		M	Antrum		√	√		
107	Krishnan	H	39822	65/M	800	√	√	M	Antrum		√	√		√
108	Abdulrahim	M	40853	65/M	800	√	√	M	Antrum	√	√	√		
109	Manivelraj	C	51286	40M	800	√	√	M	Antrum	√	√	√		
108	Perumal	H	48604	35/m	800	√	√	m	antrum	√				√
111	Cristopher	C	48670	45/M	800	√		M	Body(LC)	√		√		√
112	Mohamed bashir	M	36636	47/M	800	√	√	M	Antrum	√	√			√

94	√			√					A+ve	Antral wall thickening	Antral Growth	Adeno ca		+	Growth
95	√	√		√					B+ve	Antral wall thickening	Antral Growth	Adeno ca			Growth
96									AB+ve	Normal study	Elevated mucosa irregularity				Growt
97	√	√		√	2cm		√		B+ve	Ascites hepaticadenopathy	Antral Growth	Adeno ca			2° Live Growt Pancreas
98	√	√			3cm		√		B+Ve	Liver 2°	Growth OG jn			√	Growth no
99	√	√		√					A+ve	Antral wall thickening	Antral Growth	Adeno ca			Antral Gr infil
100		√							O+ve	Liver 2° PH nodes	Growth Body of stomach	Adeno ca			Growth stomach
101	√		√						A+ve	N	Cardia growth	Signet ring type			Growt LN,Omen
102	√	√		√	√		√		B+ve	Antral wall thickening	Antral Growth	Adeno ca			Antral
103		√		√					B+ve	Antral wall thickening	Antral Growth	Adeno ca			Antral
104	√			√					AB+ve	Distended stomach	Antral Growth				Antral
105		√							A+ve	Antral wall thickening	Antral Growth	Adeno ca			Antral C
106		√					Dvt Lt LL		A+ve	Antral wall thickening	Antral Growth	Adeno ca			Antral Gr infil
107	√		√						B+ve	Distended stomach	Growth Body			_	Growth b
108	√			√					AB+ve	Antral wall thickening	Antral Growth	Adeno ca			Antral C
109	√			√					B+ve	Antral wall thickening	Antral Growth	Adeno ca			Antral C
110	√				√		√		O+ve	N	Body of Stomach				Diffuse PD+O
111	√	√	√						B+ve	N	Antral Growth	Spindle cell ca			Grow
112		√		√					AB+ve	Antral wall thickening	Antral Growth				Antral C As

113	Sermakani	H	3927	38/F	800			M	pyloric	√	√		√	√
114	Ramar	H	16634	48/M	800	√		M	Antrum	√	√	√		√
115	Anandha naryanan	H	25798	66/M	800	√	√	M	Diffuse	√			√	√
116	Thangaraj	H	28985	42/M	800	√	√	M	Antrum	√	√			
117	Maharaja	H	32200	35/M	800	√	√	M	Antrum		√			
118	Sermadevi	H	33087	35/M	800	√		M	Antrum	√	√	√		
119	Ramalingam	H	26012	65M	800	√	√	M	Antrum	√	√		√	√
120	Thayammal	H	33860	60/F	800			M	Antral growth	√		√		√
121	Krishnan	H	39822	65/M	800			M	Antrum	√	√	√		√
122	Abdulrahim	M	40853	65/M	800	√	√	M	Antrum	√	√			√
123	Ganthimathi	H	23160	70/F	800			M	Antrum		√			
124	Arumugam	H	26615	52/M	800	√		M	Antrum	√	√	√		√
125	Perumal	H	28760	52/M	800		√	M	Antrum	√	√			
126	Murugesan	H	31863	62/M	800	√	√	M	Antrum		√	√		√
127	Ponnusamy	H	34769	60/M	800	√		M	Antrum	√				
128	Arjunan	H	3762	57/M	800	√	√	M	Antrum		√			
129	Thangalakshmi	H	43811	57/F	800			M	Antrum	√	√			√
130	Sarumathy	H	45465	40/F	800			M	Antrum	√	√	√		
131	Murugan	H	15161	31/M	800	√		M	Antrum	√	√	√		
132	Krishnavel	H	18433	31/F	800			M	Antrum	√			√	√

113	√				√		√		B+ve	Antral wall thickening	Antral Growth				Antra
114	√	√		√					B+ve	Antral wall thickening	Antral Growth				Antra
115	√		√		√		√		O+ve	Ascites	Diffuse involvement stomach	P00rly diffadenoca	√		Contract LN A
116	√			√					B+ve	Antral wall thickening	Antral Growth				Antral G
117		√		√					A+ve	Antral wall thickening	Antral Growth			_	Antral G
118	√	√		√					B+ve	Antral wall thickening	Antral Growth				Antral G
119	√	√		√					O+ve	Antral growth					Antral G
120	√	√							AB+ve	Antral wall thickening	Body and Antral Growth				Growth
121	√	√	√						O+ve	Antral wall thickening	Antral Growth	Adeno ca			Antral G
122	√		√						B+ve	Antral wall thickening	Antral Growth	Adeno ca			Antral G As
123	√		√			√			AB+ve	Antral wall thickening	Antral Growth	Adeno ca			Antral G Liver
124		√	√						A+ve	Antral wall thickening	Antral Growth	Adeno ca		+	Antra
125	√	√		√		√	√		O+ve	Ascites 2° Liver	? Linitis plastica	Adeno ca	√		Diffuse
126	√	√							A+ve	Antral wall thickening	Antral Growth	Adeno ca			Antral G
127			√						AB+ve	Antral wall thickening	Antral Growth				Antral Gr
128			√						B+ve	Antral wall thickening	Growth Body	Adeno ca			Growth B
129									A+ve	Antral wall thickening	Antral Growth	Adeno ca		+	Antra
130									O+ve	N	Antral Growth				Serosa r
131	√	√		√					O+ve	Antral wall thickening	Antral Growth	Adeno CA			Antral G
132		√							B+ve	N	Antral Growth	Adeno CA			Antral G

133	Thangathai	H	29312	50/F	800			M	Body of Stomach	√				√
134	Nallamadan	H	51675	70/m	8oo			M	Antral growth	√	√			
135	Vellammal	H	30566	70/F	800			M	Body of Stomach	√	√	√		√
136	Thangaraja	H	34538	60/M	800	√	√	M	Growth Antrum	√	√			√
137	Selvaraj	H	50590	65/m	800	√	√	M	Pyloric growth	√				√
138	Avudaithai	H	34221	45/F	800			M	Growth Antrum		√			√
139	Nainarammal	H	8591	50/F	800			M	Growth Antrum	√	√			
140	Deivakirubai	C	25414	35/F	800			M	Growth Antrum	√	√	√		√
141	Marial	c	48067	50/f	800			M	antrum	√	√	√		
142	Lashmanan	H	35426	62/f	800	√	√	M (F)	cardia	√	Dysphagia			
143	Chandran	H	7148	41/F	800				CARDIA	√	Dysphagia			
144	Jamilal	M	26987	53/F	800			FATTY MEAL	Growth Og jn		Dysphagia		√	√
145	Parwathy	H	27625	45/m	800			M	CARDIA		Dysphagia			√
146	Malliga	H	38154	38/F	800			M	OG JN		Dysphagia			√
147	Shaloni	H	28157	55/M	800			wt-69kg	OG jn		Dysphagia			
148	Rajagopal	H	38788	55/F	800			M	DIFFUSE	√			√	√
149	Selvaraj	H	51535	65/M	800	√	√	M	ANTRUM	√	√			
150	Marammal	H	44086	65/F	800			M	PYLORIC		√			√

133	√	√	√				√		AB+ve	Ascites +	Growth Body	Adeno CA			Growth B
134	√				√		√		B+ve	N	Antral Growth				Growt
135	√			√					A+ve	N	Growth Body	Adeno CA			Growth
136	√		√				√		B+ve	Antral wall thickening	Antral Growth	Adeno CA			Growth A Post
137				√					O+ve	Antral wall thickening	Antral Growth				Antral G
138		√		√					B+ve	Antral wall thickening	Antral Growth				Antral
139				√					B+ve	Antral wall thickening	Antral Growth	Adeno CA			Antral
140	√	√							O+ve	Antral wall thickening Ascites	Antral Growth	Adeno CA			Antral Gro 2°
141									A+ve	Antral wall thickening	Antral Growth	Adeno CA			Antral Gro 2°
142									A+ve	N	Cardia Growth	Adeno CA			Cardia
143									AB+ve	Ascites	Cardia Growth	Adeno CA			Growth 2° A
144									B+ve	-	Growth OG JN	Adeno CA			Oesoph
145									O+ve	N	Cardia and Fundus				Growth Fu
146									O+ve	Liver 2°	OG JN	Adeno CA			Growt Ascites
147									B+ve	N	OG JN	Poorly Diff Adeno CA			Growt Ascites
148									O+ve	Ascites	Growth Body	Poorly Diff Adeno CA			Diffuse i
149									A+ve	Antral Growth	Antral Growth	Adeno CA			Growth A
150									A+ve	Antral Growth	Antral Growth	Poorly Diff Adeno CA			Growth A

CA STOMACH PROFORMA

Name :
Age :
Sex :
Ip no :
DOA :
DOS :
DOD :
Unit :

Complaints

Mass abdomen : duration
Size
Site

Pain abdomen : duration
location
nature- colicky / dullaching /others

Jaundice : duration
obstructive/hepatocellular

Vomiting : nature food/bile/blood/others

Fever

H/o dyspepsia

H/o ball roling movements

Abdominal distension

H/o pruritis

bowel habits – constipation/diahorrea/melena/steatorrhea

H/o drug intake

Past history

DM/HT/TB/BA/IHD

H/O previous surgery

H/o jaundice

H/o blood transfusion

Personal history

Diet; veg/non veg/mixed/ fatty meals/smoked food

smoker / alcoholic

Menstrual history

Treatment history

General examination

Nutrition

Hydration status

Anaemia.

Jaundice.

Pedal edema.

Lymph nodes.

Vitals-PR : BP : RR : TEMP :

SYSTEMIC EXAMINATION:

PER ABDOMEN :

INSPECTION :

Site :

Size :

Shape :

Umbilicus :

Veins/pulsation

Moves with respiration.

PALPATION:

Consistency

Warmth tederness

Guarding/rigidity

Organomegaly

PERCUSSION:

Liver dullness

Over the mass

Free fluid

Shifting dullness

Fluid thrill

AUSCULTATION

Bowel sounds

DRE

OTHER SYSTEMS - CVS/RS/CNS/LOCOMOTOR

CLINICAL DIAGNOSIS

INVESTIGATIONS

HB%

TC

DC

ESR

URINE - alb/sug /dep

BLOOD - sug/urea/creatinine

SR - electrolytes-na,k

LFT - sr.bilurubin

SGOT/AST

SGPT/ALT

Sr.proteins - alb/glo/total

X RAY CHEST PA VIEW

XRAY ABDOMEN - supine/ erect

Endoscopy - upper gi endoscopy

HPE Report

H.PYLORI

OPERATION NOTES

POST OP FOLLOW UP

Day of ryles tube removal

Day of suture removal

Complications

Period of follow up - RT/CT

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Profile of 150 Cases of Carcinoma Stomach in Tirunelveli Medical College Hospital

DISSERTATION SUBMITTED FOR

M.S. GENERAL SURGERY

DEGREE EXAMINIATION

TIRUNELVELI MEDICAL COLLEGE HOSPITAL

TIRUNELVELI.

MARCH - 2009

THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY,

CHENNAI, TAMILNADU

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CONTENTS

INTRODUCTION

Carcinoma stomach is one of the leading causes of morbidity and mortality accounting for 12% of deaths. There has been a decline in its incidence since 1930. Even though the incidence of distal gastric cancer is decreasing in western countries, incidence of proximal gastric cancers continue to increase.

The overall 5-year survival rates ranging from 53% in Japan to 10% in Eastern Europe.A better outcome is obtained in Japan due to an active screening programme resulting in earlier diagnosis and an aggressive surgical approach. The combination of chemoradiation therapy and curative resection increases disease free and overall survival duration which has led to recommend multimodality treatment in patient with systemic disease. Inspite of this multimodality treatment the morbidity and mortality continues to be high.

AIMS OF THE STUDY

This study was mainly to analyse cases of carcinoma stomach in our region which commonly present to the general surgeon in the department of general surgery , Tirunelveli medical college hospital, Tirunelveli.

The study was conducted in the following principles.

- To study the anatomical location of growth with respect to age and gender distribution
- To study the Etiology & Risk factors with respect to anatomical location.
- Study of H.Pylori association with carcinoma stomach.
- To study the sensitivity and specificity of various investigation modalities used.
- To study the Histopathological variety in relation to the site of growth
- To study the mortality and morbidity rate with reference to treatment modality.

MATERIALS AND METHODS

This study was conducted in the general surgery department , Tirunelveli medical college hospital ,Tirunelveli for a period of 30 months, from June 2006 to November 2008.

- 150 Cases of Carcinoma stomach were studied during this period.

- Details of 150 patients with Adenocarcinoma were retrieved and follow up of these patients was done with the surgical treatment modalities used and its outcome and survival.

- Cancer recurrence after surgery and cancers after GJ for peptic ulcer disease were excluded from this study.

- Age, gender, predisposing factors, clinical presentation and surgical treatment modalities used were recorded in a prestructured proforma.

- The site of the stomach tumours was recognized as involving one of the four sites. Namely

  1. OG Junction 2. Proximal Stomach (Cardia & Fundus)

  3. Body 4. Antrum

- Only those cases which received some form of surgical treatment were included in the study.

- Association of H.Pylori with gastric cancer study was carried out in eleven patients of histologically proved gastric carcinoma.

- The WHO classification of HPE report was combined with Lauren's HPE type in order to study the Histopalthology type in relation to tumour location.

DATA ANALYSIS

- The Commonest abdominal malignancy in our region is CA stomach.
- The Common site of involvement in CA stomach is Pyloric Antrum constituting 117 out of 150 cases.
- This was in concordence with the study of cherian et al (2007), Sivaraman et al.
- This study, is contradictory to the rising incidence of AdenoCA of proximal growth in the West, Iran, Sweden & Kerala

SITE DISTRIBUTION

Site	No of cases	%	1993, TVMCH
OG jn	6	4%	2%
cardia	7	4.6%	2%
Body	15	10%	24%
pylorus	117	78%	48%
Linitisplastica	5	3.3%	10%

- Proximal growth shows an increase of 2% when compared to the previous study.
- A decline in the incidence of body growth (10%) was found in this study.
- Antral growth continues to increase by 30% in this study.
- The study revealed that the distal growth is continue to be increasing our region than the proximal growth .

AGE DISTRIBUTION

Site	Age groups				
	<30	30-39	40-49	50-59	>60

OG jn			2	2	2
Cardia			2	3	2
Body		2	7	2	4
antrum	1	8	16	34	52
Linitisplastica			3		2

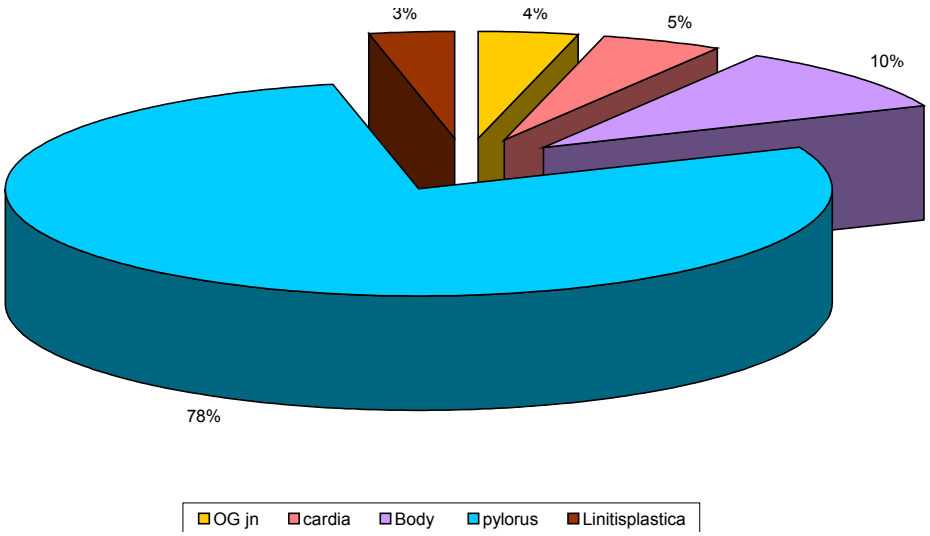
- The age group affected is evenly distributed in cancers involving OG jn above 40 years..

- Significant increase in the number of patients after the age of 50 years was seen in cancers involving pyloric antrum.

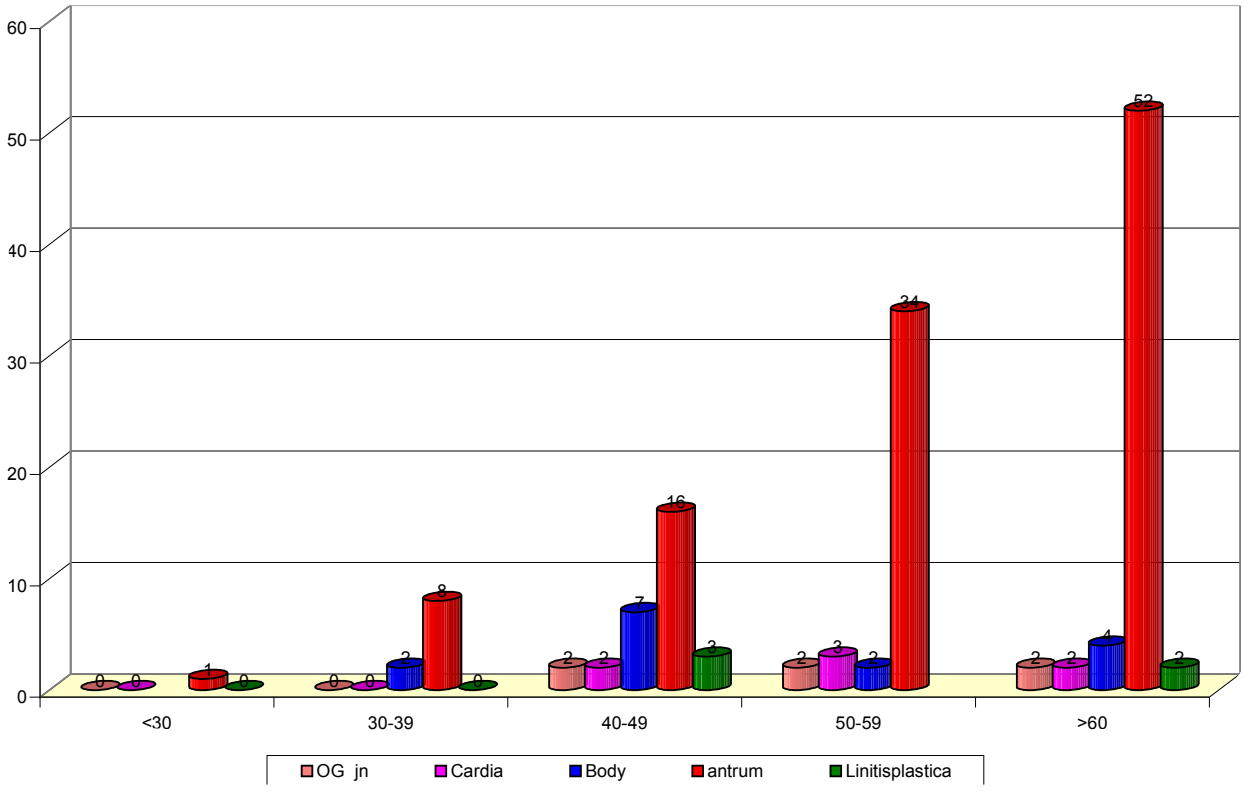
- Both the youngest age(24yrs) at presentation and oldest age(84yrs) of presentation were seen in Antral growth.

- This is concordance with the results of Lee et al and Kim et al.

SITE DISTRIBUTION



AGE DISTRIBUTION



GENDER DISTRIBUTION

Site	Male	Female	Ratio
OG jn	3	3	1
Cardia and fundus	6	1	6
Body	11	4	1.2
Pyloric antrum	96	20	4.6
Linitis plastica	3	2	1.5

- The male : female ratio of cancers involving the proximal stomach, body, antrum and OG Jn was 6, 1.2, 4.6 and 1 respectively.
- This was not in concardance with the study of Cherian et al, where the ratio is 3, 2, 3 and 4 respectively.

MEAN AGE DISTRIBUTION

Site	Male	Female	SJE 2007 Male	SJE 2007 Female
OG jn	47.5	47	50.5	46.25
Cardia&fundus	51.4	45	55.47	50
Body	52.5	58.1	55.43	46.37
Antrum	56.7	60.1	55.5	52.86
Linitis plastica	51.3	57.5	-	-

- Mean Age of occurrence is low in both sexes having proximal growths.
- This is in concardance with the study of Saudi gastroenterology 2007.

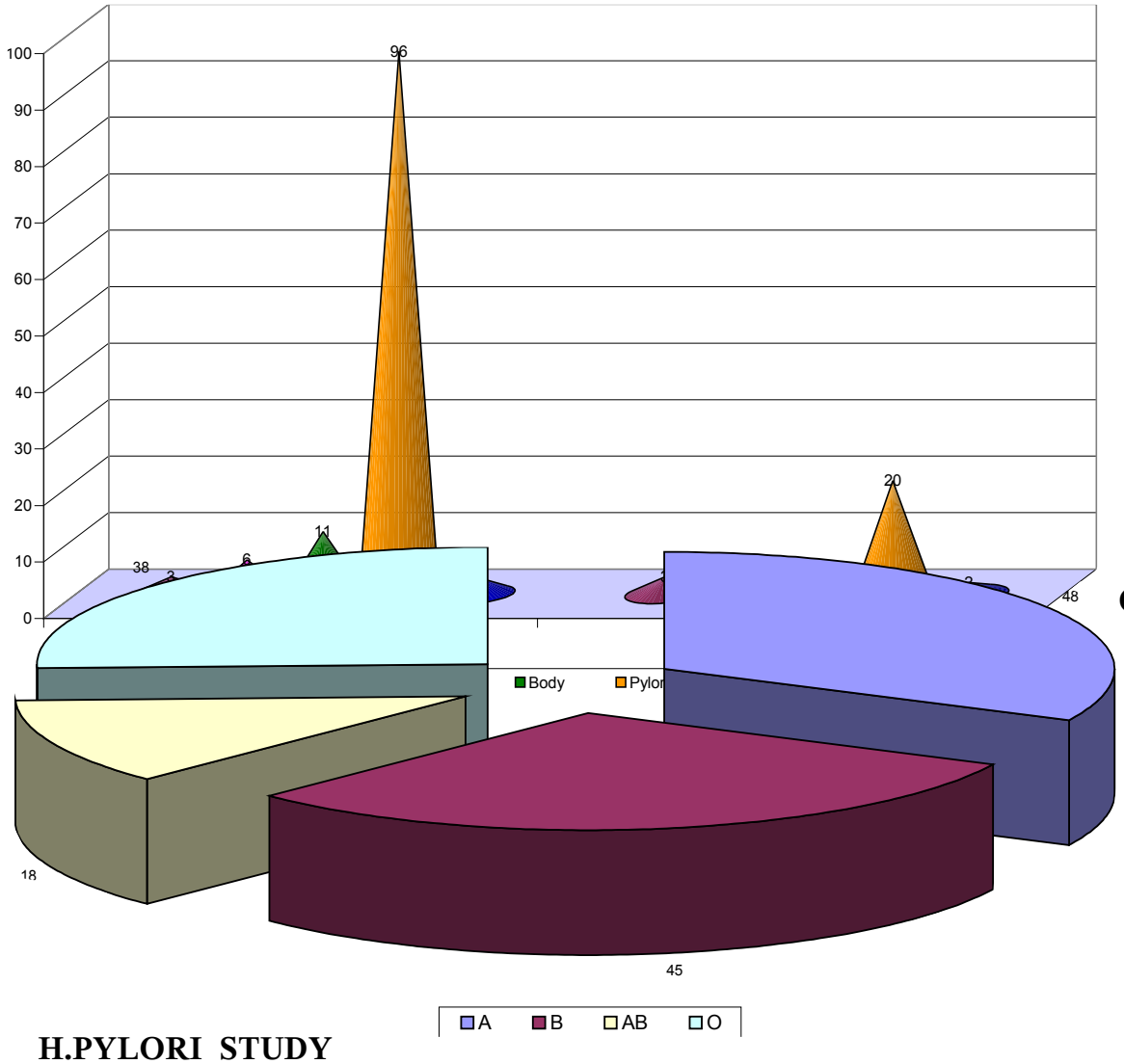
ETIOLOGICAL ASSOCIATION

Site	Smoking	Alchohol	Smoked, spicy foods	Blood Grouping				Fatty food
				A	B	AB	O	
OG jn	3	1	2	2	3	0	1	6
Cardia&fundus	3	1	1	3	0	2	1	7
Body	3	2	12	2	3	4	6	5

Antrum	62	48	78	39	39	12	27	15
Linitis plastica	3	1	2	2	0	0	3	1

- Smoking contributes 100% association of cancers in OG Jn in males and 71.4% in cardia and 64.58% in Antral growth.
- Gammon et al, Inoue et al describes the association between smoking and proximal gastric cancers.
- Blood group A is the most common group affected by Carcinoma Stomach in our region. AIRD describes the association between ca stomach and Blood group A.
- Smoked and spicy food shows an increased association with antral growth.
- Fatty food has an increased association with proximal cancers.

GENDER DISTRIBUTION



BLOOD GROUPING

H.PYLORI STUDY

- 11 Patients underwent study of H.pylori by subjecting them to ELISA of IgG Antibody of H.Pylori kit
- About 5ml of blood was drawn from each patient,serum was separated and the IgG Antibodies were measured.
- The results were interpreted as positive if the value is above 20IU and negative if it

is below 20IU.

- One patient with OG jn growth ,two patients with body of growth, eight patients with pyloric antral growth were subjected to study.

- The study includes seven patients of more than 60 years of age, two patients of fifty to sixty years of age, one patient below 50 years of age and one patient of thirty five years of age.

- Of these six patients are with blood group A, eight patients with intestinal type of lauren's classification, one patient with mucinous Adenocarcinoma and two patients with poorly differentiated Adenocarcinoma.

From this study ,the following results were obtained

- H. Pylori showed an association with intestinal type of 86.5%

- This is in concordance with study of Anderson et al of 90%

- Age group affected in H.pylori infection is above 60 yrs.

- This study corelates well with study of Feilding et al & Hunt et al, shows an association of age group affected above 60 yrs.

- H. Pylori shows an association with antral growth in 27.2% of patients and body of the

stomach in 18% of patients

- Sibata et al shows an association of 68% with antral growth 50% with cardia growth and 66% with body of growth.

- H.Pylori shows an 50% Association with BLOOD Group A.

SENSITIVITY & SPECIFICITY OF INVESTIGATIONS

Site	Endoscopy	USG	BA meal	Ba swallow
OG jn	96%	0%	-	42.8%
Cardia&fundus	100%	0%	-	
Body	100%	20%	-	-
antrum	96.39%	90%	-	-
Linitis plastica	20%	40%	100%	-

- Endoscopy was done for 140 cases
- Endoscopy and Biopsy shows 96.39% in detecting Antral growth and 100% in detecting Cardia & Body growth and 96% in detecting growth in OG Jn.
- USG has a sensitivity in detecting Antral growth as Antral wall thickening in 90% of cases.
- Ba meal has a sensitivity of 100% in detecting Linitis Plastica.

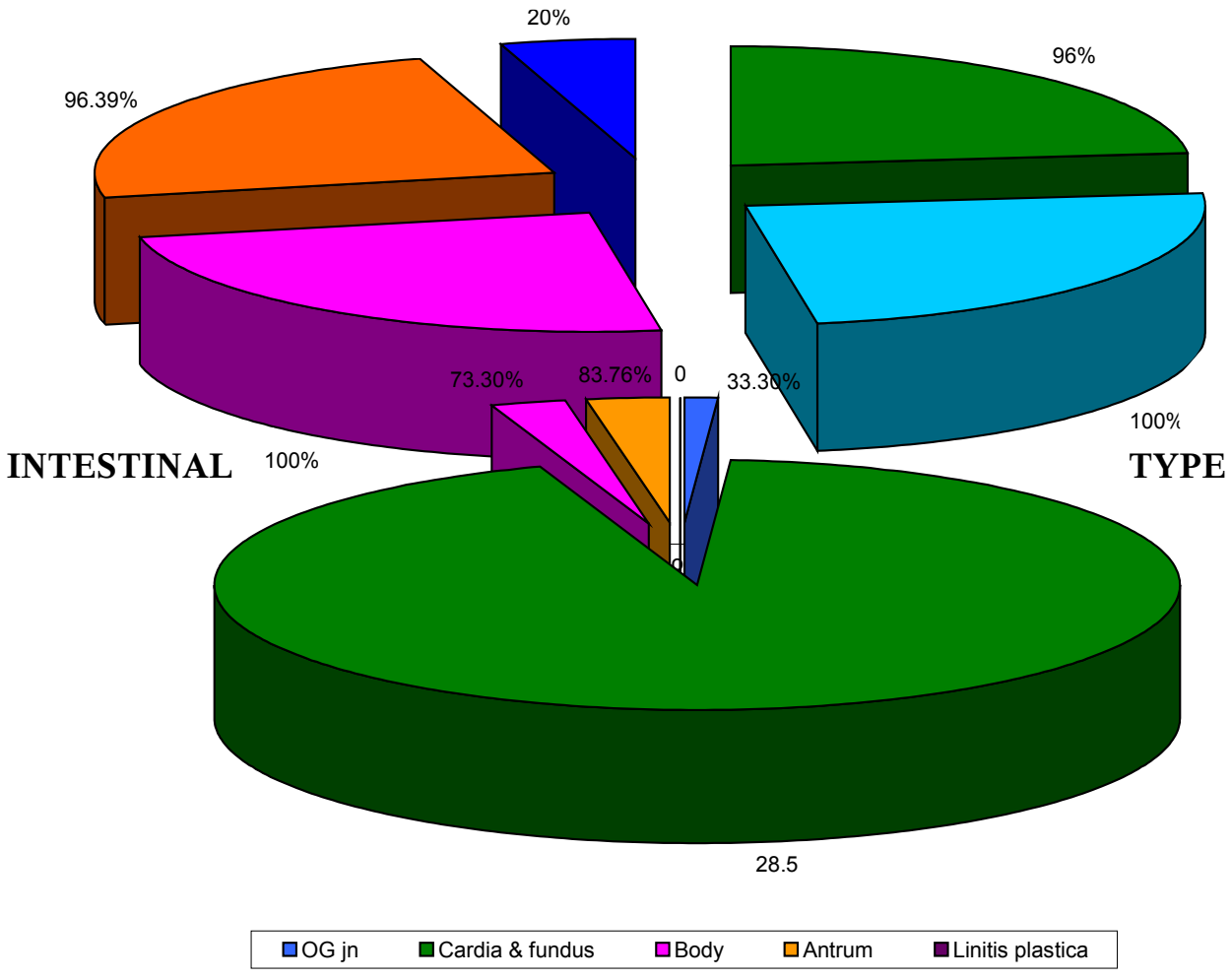
HISTOPATHOLOGY ASSOCIATION

Site	Intestinal type	Diffuse type	Mixed	Chandha, khan et al 2007		
				Intestinal type	Diffuse type	Mixed type
OG jn	2(33.3%)	4(66.6%)	-	-	-	-
Cardia & fundus	2(28.5)	3(66.6%)	2	5.3%	15.3%	-
Body	11(73.3%)	4(2.6%)	-	8%	15.3%	4%

Antrum	107 (91.45%)	7(5.9%)	3	28%	27.3%	8%
Linitis plastica	-	5(100%)	-	-	-	-

- The Intestinal type of Lauren's classification is found in 91.45% of cases of Antral Growth.
- The diffuse type contributes to 5.9% in growth of Antral cancers.
- The mixed type contributes 1% in Antral growth.
- The diffuse type is predominantly seen in proximal cancers and Linitis Plastica contributing 66.6%.

ENDOSCOPY



Chandha et al and khan et al in 2007 describes intestinal type is more common in the pyloric antrum and cardia and fundus growth shows a predominance of diffuse type.

STAGING

Stage	No of patients	%
Stage 1a	-	-
Stage 1b	-	-
Stage 2	10	6.6%
Stage3a	11	7.3%
Stage3b	3	2%
Stage 4	126	84%

Site	Stage 1a	Stage 1b	Stage2	Stage3a	Stage3b	Stage4
OGjn			1			5(83.3%)
Cardia			1	1		5
Body			2	1	1	11
Antrum			6	9	2	100(66.6%)
Linitis						5(100%)

- The proximal tumours are usually advanced at presentation and have poorer long term prognosis -anderson et al

- In this study OG jn growth shows of 83.3% STAGE 4 disease &pyloric antrum shows 66.6% of STAGE 4 disease

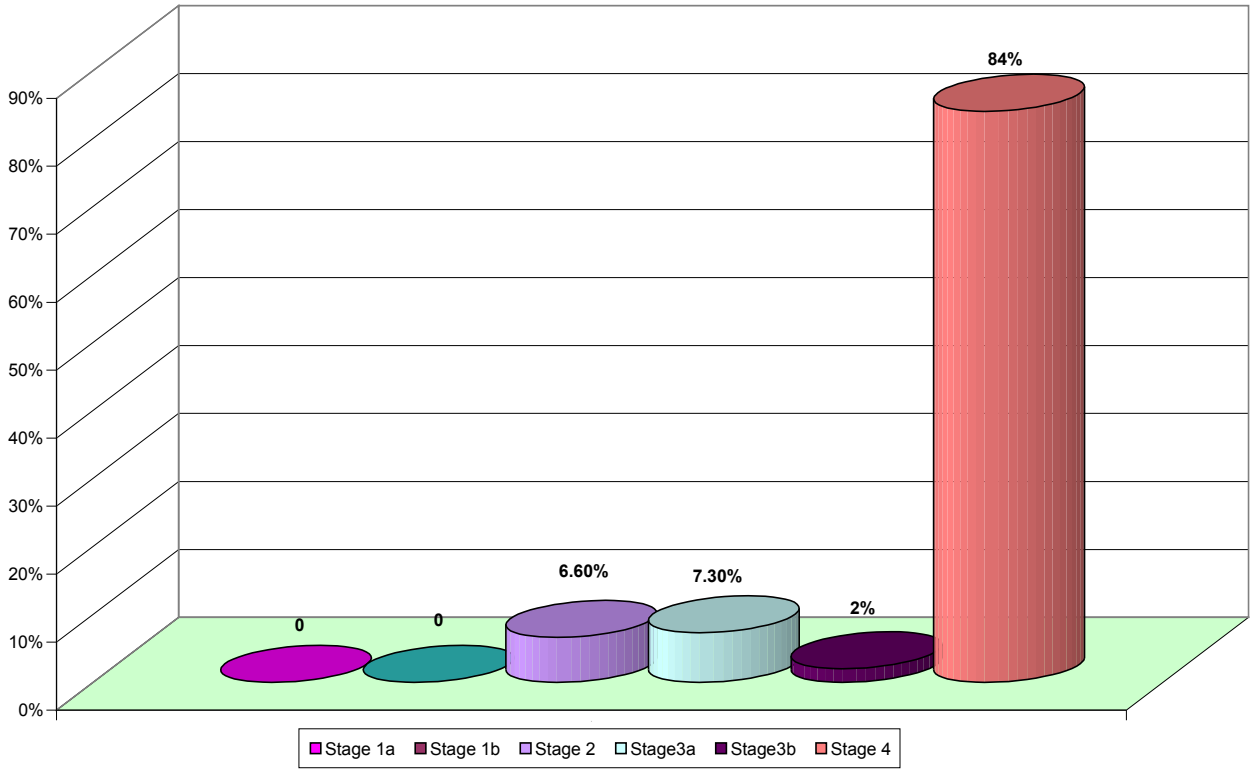
NODAL STAGING

Staging	No of patients	%
No	28	18.6%
N1	94	62.6%
N2	20	13.3%
N3	1	0.6%

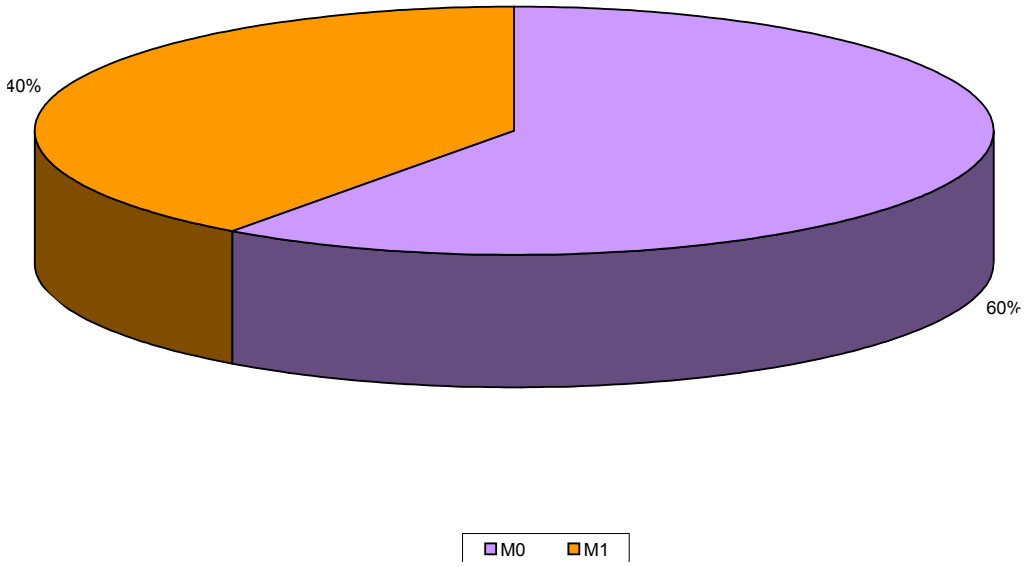
M STAGING

Staging	No of patients	%
M0	24	16%
M1	16	10.6%

STAGING



M STAGING



SURGICAL RESULTS

Surgery	No of cases	Morbidity	Mortality	Mortality
Total gastrectomy	8	12.5%	37.5%	8%
Sub total gastrectomy	8	-	-	10%
Distal gastrectomy	10	-		-
Distal gastrectomy and Roux en y	3	-	-	-
Palliative distal gastrectomy	44	-	2.8%	7%
Anteriorgastrojejunostomy	63	1.3%	9.5%	-
Feeding jejunostomy	14	-	2.8%	-

- Total gastrectomy was done for eight patients with a morbidity rate in two cases and mortality of three cases.
- The palliative procedure shows high morbidity may be due to disease process in my study.
- Anastomotic leak was found in six patients with 4%.
- .The most devastating complication is anastomatic leak in 3%-21% of cases - anderson et al
- Death due to co morbid illness in 4% of patients.
- Sub total gastrectomy and curative distal gastrectomy did not show any mortality in my study.

REVIEW OF LITERATURE

Carcinoma stomach is the one of the leading cause of cancer related death in world wide. It is the eighth most common cancer in US. It is the seventh most common cancer In India. There has been decline in its incidence since 1930. Although the incidence of distal gastric cancer is decreasing in the world, the incidence of proximal gastric tumors continues to increase.

The approximate incidence of gastric carcinoma in the United States is 10cases/1,00,000 people. In Japan -78 Cases/1,00,000 people. In India, cancer stomach is the 6th common cancer in male and 7th most common cancer in female.In TVMCH the incidence of ca stomach is 8.79% based on cancer registry during the period 2001-2002. In particular, gastric cardiac tumors are five times more common and non-cardia gastric tumors are twice as common in men as women. The incidence is also higher in low socio economic populations of developing countries. Survival rates in patients with gastric cancer is poor. The overall 5-year survival rates ranging from 53% in Japan to 10% in Eastern Europe. The analysis of the national cancer Data base of US revealed 6% to 12% better 5 year survival rate for women than for men, for patients with distal as opposed to proximal tumours. continues to be high.

HISTORY

Billroth, Wölfler	1881	First successful resection on human patient
Wölfler,nicolondi	1881	Performed antecolic gastrojejunostomy

Braun	1893	Described jejunojejunostomy as routine addition to gastrojejunostomy
Billroth,	1874	Billroth I - Distal gastric resection with anastomosis of Stomach and duodenum. Billroth II - Anastomosis of stomach with jejunum through transverse mesocolon

ANATOMY OF STOMACH.

For descriptive purposes ,the stomach is divided into

Lesser curvature

This continue with the right free border esophagus and forms the concave border of the stomach.

Greater curvature

This starts at the cardiac notch and forms the convex border of the stomach.

The fundus

This is the area above the horizontal line from the cardiac notch to the greater curvature of the stomach.

The incisura angularis

In a J shaped stomach this is the junction of vertical and horizontal parts of the lesser curvature

The body

This is the portion of stomach lying between the fundus and pyloric portion of stomach.

The pyloric portion

The distal one fifth of the stomach consists of pyloric antrum ,canal and sphincter .The pyloro duodenal junction is identified by the vein of mayo.

SURGICAL ANATOMY OF STOMACH

SKANDALAKIS ET AL divide the stomach into proximal gastric unit

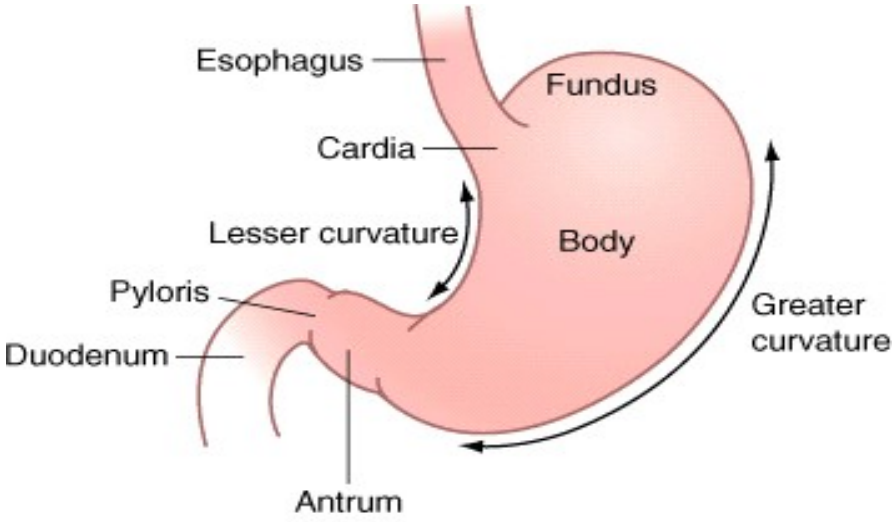
and distal gastric unit.

Proximal gastric unit consists of

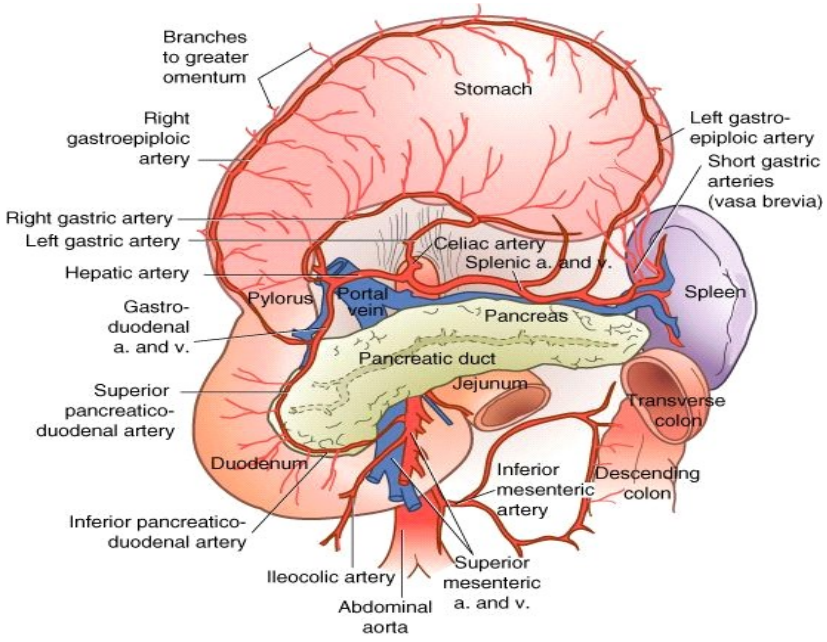
Distal esophagus - 0.5 -2.5cm

Og jn and esophageal hiatus

ANATOMY OF STOMACH



BLOOD SUPPLY OF STOMACH



Proximal stomach(cardia &fundus)

Distal gastric unit consists of

Gastric antrum

Pylorus

1st part of duodenum -first 2.5 cm of duodenum

Relations of proximal gastric unit

Lesser & greater curvature

The upper part of lesser sac

OG jn

Lesser curvature of body, GE jn and the abdominal esophagus are attached to the hepato gastric ligament and its contents .The greater curvature attaches to the upper part of greater omentum and several related splenic ligaments.like gastrosplenic and splenoleinal ligament.

Relations of distal gastric unit

The lesser curvature of antrum, pylorus and upper part of duodenum are attached to hepatogastric and hepato duodenal ligament

The greater curvature is attached to gastrocolic ligament.

Posterior relations of distal gastric unit

- Floor of lesser sac
- Transverse mesocolon
- head & neck of pancreas

- Aorta&celiac trunk & branches
- Celiac ganglion & plexus
- Hepatic triad
- Gastro duodenal artery

Anterior relations of distal gastric unit

- Anterior abdominal wall
- Medial segment of left lobe and anterior segment of right lobe of liver
- Transverse mesocolon
- Neck of gall bladder

BLOOD SUPPLY OF STOMACH

The four main arteries of stomach are

LEFT GASTRIC ARTERY

Arises from coeliac axis The left gastric artery commonly divides into an anterior and a posterior branch before attaining the lesser curvature[111] and, in such cases, the esophageal and cardioesophageal arteries may arise, variably, from either of those vessels; more commonly, however, the cardioesophageal artery arises from the anterior gastric branch.

After its cardioesophageal branch the left gastric artery usually curves downward to the

right and thereafter descends along the lesser curvature. As it descends, it bifurcates into an anterior branch which sends branches to the anterior gastric wall, and a posterior branch which, similarly, supplies the posterior gastric wall. A smaller continuation of the left gastric artery continued along the lesser curvature.

RIGHT GASTRIC ARTERY

The right gastric artery is a small branch which arises most commonly from the proper hepatic artery (50-68%), left hepatic artery (28.8-40.5%), common hepatic (3.2%), The right gastric artery gives origin to one or more suprapyloric branches. Anterior and posterior branches from these anastomose with infrapyloric vessels and with the supraduodenal artery, providing for the distal gastric unit (antrum, pyloric canal, first inch (2.54 cm) of the first part of the duodenum). The right gastric passes along the lesser curvature for about 4 to 6 cm, about 0.5 cm from the lesser curvature, before anastomosing with the left gastric. In about 13% of individuals, the right gastric artery provides origin for the supraduodenal artery.

RIGHT GASTRO EPIPLOIC ARTERY

The right gastroepiploic artery is a branch of the gastroduodenal artery (or its continuation) in most cases. It occasionally arises from the superior mesenteric artery or from the anterior superior pancreaticoduodenal artery. After giving origin to an infrapyloric branch, the artery passes along the greater curvature of the distal gastric surgical unit within the gastrocolic ligament. It gives origin to 8-18 singular or paired anterior and posterior branches for the gastric wall. The gastric branches of the right gastroepiploic artery pass mostly undivided in a submucosal position about one-fifth of the distance from the greater curvature.

They anastomose extensively with branches from the left gastric artery.[108] If the first infrapyloric branch is particularly large, the first following gastric branch tends to take its origin further around the greater curvature. In about 75% of individuals, the right gastroepiploic artery clearly anastomoses with the left gastroepiploic,[111] although not so profusely as is seen in the anastomoses with the left gastric artery.

The gastroduodenal artery ran in front of the common bile duct and descended along the posterior surface of the head of the pancreas

LEFT GASTRO EPIPLOIC ARTERY

The left gastroepiploic artery is the largest branch of the splenic artery.[56] It reaches the stomach at a point about halfway along the greater curvature, after it has already delivered several rather long branches to the more proximal part of the greater curvature. These branches reach the stomach by way of the gastrosplenic ligament.The left gastroepiploic artery gives off the left epiploic and the anterior epiploics. Together with similar branches of the right gastroepiploic, they form the arc of Barkow , which is reinforced also by the posterior epiploic branches of the inferior pancreatic artery .

VEINS OF THE STOMACH:

The veins of the stomach mainly accompany the arteries. Of particular importance is the left gastric or coronary vein,receives branches from the esophagus.

LYMPHATIC SUPPLY OF STOMACH

The lymph nodes concerned in the drainage of stomach are.

HEPATIC GROUP

Lymph nodes lie in the lesser omentum along the bile ducts and receive lymph from the liver and gall bladder.

SUB PYLORIC GROUP

Lie in the angle between first and 2nd parts of duodenum on the head of pancreas in relation to duodenal artery.Receive the lymph from right two thirds of greater curvature of stomach.

GASTRIC GROUP

Superior nodes - lie along left gastric artery and para cardial nodes

Inferior nodes - lie along the pyloric half of greater curvature of the stomach

PANCREATICOLIENAL GROUP

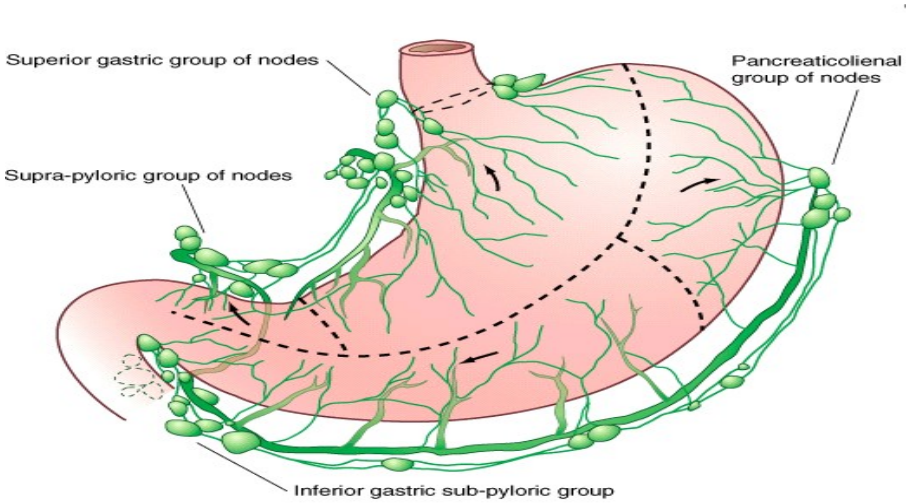
These lie along the splenic artery in relation to upper border of pancreas.

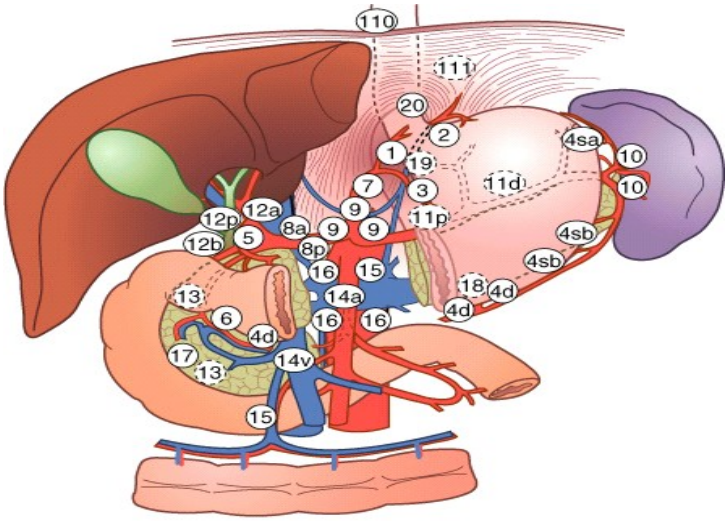
JAPANESE CLASSIFICATION OF LYMPH NODES

1. Rt. Paracardial

2. Lt Paracardial
3. Along Lesser Curve
4. Along Greater Curve
5. Suprapyloric
6. Subpyloric
7. Lt Gastric
8. Common HepaticCoeliac Axis
9. Splenic Hilum
10. Splenic Artery
11. Hepatoduodenal Ligament
12. Retropancreatic
13. Superior mesentric

LYMPHATIC SUPPLY OF STOMACH





JAPANESE CLASSIFICATION OF LYMPHATIC SUPPLY OF STOMACH

14. Middle colic
15. Para aortic
16. Anterior pancreatic
17. Inferior pancreatic
18. Infra diaphragmatic
19. Oesophagial Hiatus
20. Lower Paraoesophageal
21. Supra diaphragmatic

The gastric wall consists of the serosa, the muscular layer, the submucosal layer, and the mucosal layer.

SEROSAL LAYER

The serosa is nothing more than the peritoneum, a thin layer of loose connective tissue underlying a layer of simple squamous mesothelium.

MUSCULAR LAYER

The anatomist describes the three well-known layers of musculature: an outer longitudinal layer, middle circular layer, and inner oblique The outer longitudinal layer of muscle is present along the lesser and greater curvatures Its fibers are arranged into two groups. The first set is continuous with the outer smooth muscle layer of the esophagus. The fibers are best developed along the curvatures, ending proximal to the pyloric region. The second set begins in the body and passes to the right, with the fibers becoming thicker as they near the pylorus.

The middle circular layer rather uniformly encircles the entire stomach and "is the principal part of the muscular coat," according to Woodburne.[55] This layer becomes thicker at the pylorus, most distally forming the pyloric sphincter .

The inner oblique muscle layer, internal to the circular layer, is limited to the body of the stomach and is most developed near the cardiac orifice.

SUBMUCOSAL LAYER

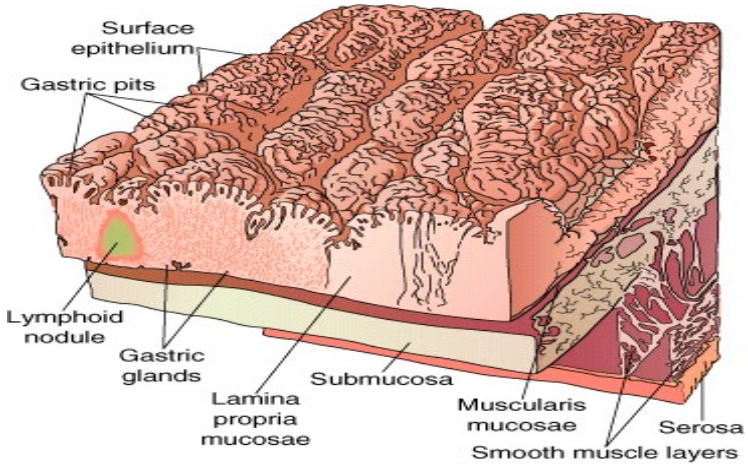
The submucosa is composed of loose, areolar connective tissue which connects the mucosa to the external musculature. It can be called the "vascular layer" (both arterial and

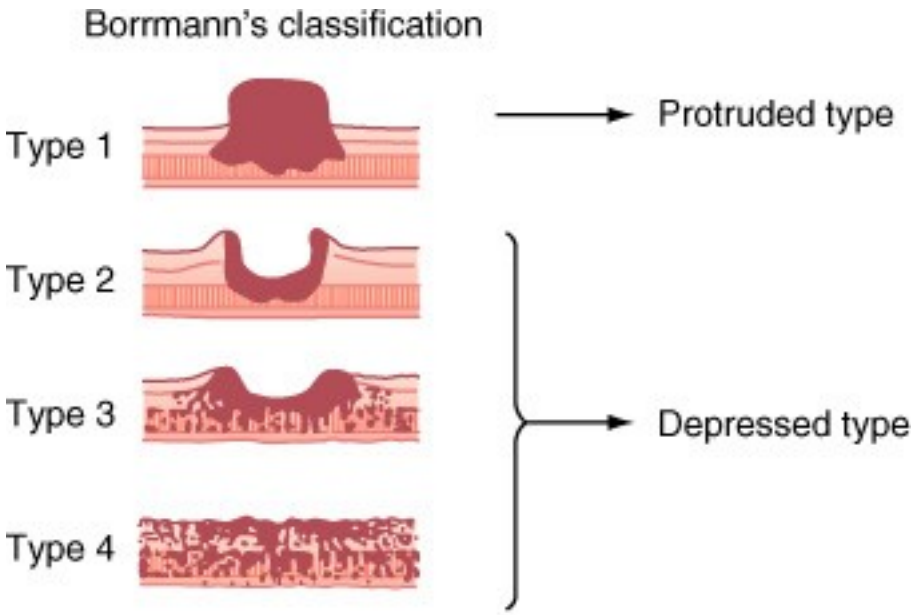
venous) of the gastric wall. It houses plexuses that have rich anastomoses through extensive ramifications that form excellent collateral flow. All the parts of the gastric mucosa receive blood from th ese submucosal plexuses except the lesser curvature.

MUCOSAL LAYER

- The distal esophagus is lined by stratified squamous epithelium, with mucous cells in the abdominal esophagus
- The cardia contains simple columnar cells

GASTRIC MUCOSA SURFACE





- The fundus and body contain two types of cells: parietal (oxyntic) acid-secreting cells and chief pepsin-secreting cells
- The lamina propria of the fundus and body is filled with branched, tubular gastric glands

RISK FACTOR

HELICOBACTER . PYLORI

Sibata et al & Huang et al found H.Pylori positivity is more associated with non-cardia gastric cancer. H.plyori was detected in 90% of intestinal type of cancer &32% of diffuse type cancer. It is also as associated with MALT and gastric Lymphoma. H.Pylori increases the risk of gastric cancer by impairing the bioavailability of vitamin C.

WARREN, in 1980 detected a curved s shaped bacilli in light microscopy. Initially it was recognized as campylobacter jejuni.in October 1989, the organisms was named helicobacter, because of the presence of sheathed flegelle,a unique fatty acid profile, different repiratory quionones.The isolation of H.Pylori from the gastric mucosa and the report of the organisms urease activity was postulated by MARSHALL.

H. Pylori is almost associated with inflammatory response.The spiral shaped organisms allows efficient motility in the mucus and in the gastric juice. The enzyme urease by breaking down urea in the gastric juice appears to generate enough bicarbonate and ammonium ions around the organisms to allow its safe passage through the gastric acid barrier to reach the mucous layer.Ammonia elevates the pH of the gastric mucosa from about 6-7.It is known to deplete aerobic cells of alpha keto glutarate, an essential substrate for the TCA acid cycle. Once attached to the mucus layer and the mucosa, H.Pylori, secretes soluble proteases and

phospolipase, which may be harmful to the mucus layer.

The vacuolating cytotoxin is a 87 kda protein , expressed in about 65% of H.Pylori strains and is responsible for creating vacuoles in the epithelial cells. The gene for cytotoxin protein is V ac A. The Vac gene is present in all H.Pylori bacteria but only produces an active protein in 65% of isolates.A second protein at 127 kda is called cytotoxin associated gene and is called Cag A gene. Cag A gene is a marker for vacuolating cytotoxin effect and is only present when Vac A cytotoxin is present. The organisms have been classified into Type I 0rganisms which have Cag A and Vac A which are more ulcerogenic and type II organisms that lack Cag A and do not produce cytotoxins. H. Pylori infection is associated with increase gastrin levels and decrease somatostatin levels, which has led to the development of atrophic gastritis ,a precancerous condition of gastric carcinoma. Epidemiological studies have demonstrate that there is an association of H.Pylori with gastric carcinoma . this infection has three fold to six fold higher risk for developing gastric cancer than those without infection.

The incidence of gastric carcinoma tends to be high in geographic regions were people consumes diets high in salt & smoked foods. The presence of nitrosamines in the smoked food is a carcinogen in developing the carcinoma.

Smoking has a five-fold increased risk of cardia and upper-third gastric cancer and a two-fold increased risk of distal gastric cancer compared to nonusers.

Male gender, Black race & low socio economic class are associated with Gastric

carcinoma.

Obesity is associated with Proximal gastric cancer.

Occupational hazard exists for metal workers is a associated factor for distal gastric cancer..

An associate between gastric carcinoma and blood group A was described in 1953 by…AIRD ET AL….

PREDISPOSING FACTORS

Gastric polyps particularly Villous Adenomas indicate increased risk of malignancy.

Pernicious Anemia is associated with 10% incidence of gastric cancer.

Mutations in CDH1…. Gene that encodes **E. Cadherin** is more common in diffuse type gastric cancer. Mutations in the CDH1 gene that encodes E-cadherin, an epithelial cell adhesion molecule, may be found in intestinal cancers but are more common in diffuse-type gastric cancers. Furthermore, defects in E-cadherin mediated cell adhesion are characteristic of diffuse-type gastric tumors. It also appears that a CDH1 gene mutation occurs in a cluster of patients with hereditary diffuse gastric cancer. Therefore, prophylactic gastrectomy is offered to carriers of these mutations. Virtually all carriers of this mutation so far have been found to harbor an early malignancy in the resected specimen, despite negative initial endoscopy findings, pointing to the advisability of the gastrectomy.

Vascular endothelial growth factor C (VEGF-C) is a glycoprotein that belongs to the VEGF family. VEGFs are cytokines that play an important role in angiogenesis. In gastric adenocarcinomas, expression of VEGF-C mRNA is associated with lymphatic invasion, lymph node metastasis, and possibly a less favorable outcome

P53 over expression revealed in more than 55% of tumors.

Previous partial gastrectomy & **Menetrier's disease** – Pre disposing factor for gastric cancer.

Hypogammaglobulinaemia is associated with gastric cancer.

PATHOLOGY

Approximately 10% of these tumours are polypoid fungating tumours, with a relatively good prognosis after aggressive surgical management. **Ulcerating or penetrating cancers,** which occur in more than 50% 0f patients, are sessile. **Superficial spreading carcinoma** is more unusual(6% of tumours). This tumour is diffusely infiltrative over a wide area,although it is predominantly confined to mucosa and submucosa. A further subgroup of diffusely infiltrative cancer is the LINITIS PLASTICA (leather bottle) carcinoma and carries particularly poor prognosis.

EARLY GASTRIC CANCER

The term early gastric cancer describes tumours confined to the gastric mucosa

and submucosa, irrespective of nodal status. Based on endoscopic appearance,(JAPANESE CLASSIFICATION),it is divided into three groups,type-1 protruding ,type -2 superficial ,and type-3 – excavated. Type -2 is divided into three subgroups,a) elevated b) flat and c)depressed Early gastric cancer accounts for 40% of all gastric cancers in Japan. In the west,early gastric cancer accounts for 9% of all cancers detected.50% of cases progress to advanced cancer within three years from the time of endoscopic diagnosis, and almost all patients develop advanced disease within five years.

CA STOMACH WITH ASCITES

METASTATIC CUTANEOUS NODULES IN CA STOMACH

ADVANCED GASTRIC CANCER

Advanced gastric cancer suggests invasion of the muscularis mucosa or beyond. It shows serosal involvement, invasion of adjacent organs,extensive nodal metastasis and secondary deposits usually within the peritoneal cavity.By, definition these tumours always exceed two cm in diameter (.BORRMANS CLASSIFICATION)

MICROSCOPIC APPEARANCE

The great majority of gastric cancers are Adenocarcinoma; other tumours are rare.

WHO CLASSIFICATION

The WHO classification based on morphology divides gastric cancers are into five types:adeno carcinoma,adenosquamous carcinoma, squamous cell carcinoma, undifferentiated carcinoma, and unclassified carcinoma. Adenocarcinomas contribute 95% of gastric cancers which arises from the mucous producing rather than acid producing cells of gastric mucosa The adeno carcinoma are divided into four patterns; papillary, tubular, mucinous, and signet ring, which may be divided by degrees of differentiation. In tumours presenting a mixed picture with varying degrees of differentiation in the same tumour ,is based on the predominant type.

Lymphomas , carcinoid, Leiomyosarcoma, GIST of stomach , adenosquanmous and squamous cell carcinoma comprise 5% of gastric cancers.

LAUREN'S CLASSIFICATION:

According to Lauren's classification, the two Histologic types of gastric carcinoma are intestinal and diffuse. The intertinal type have the tendency of Malignant cells to form glands. The diffuse type lacks organized gland formation, ie usually poorly differentiated and has many signet ring cells.The intestinal type carries the the more favorable prognosis and is more common in areas with a high incidence of the disease.

MING CLASSIFICATION

In this classification ,cancers were divided into an expanding type that produces nodules that compress adjacent tissue (Laurens intetinal type), and the infiltrative type that does not forms masses(Lauren's diffuse type)

CLINICAL PRESENTATION

Gastric adenocarcinoma is usually not associated with specific symptoms early in the course of the disease. Patients often ignore the vague epigastric discomfort and indigestion that portend the cancer and may be treated presumptively for benign disease for 6 to 12 months before diagnostic studies are performed. Rapid weight loss, anorexia, and vomiting are usually a sign of advanced disease. These presenting features are simply due to the presence of a partially obstructing lesion .

The epigastric pain is usually similar to the pain caused by benign ulcers and is often relieved by eating food; however, it can mimic angina. Dysphagia is usually associated with

tumors of the cardia or gastroesophageal junction. Antral tumors may cause symptoms of gastric outlet obstruction. Although very rare, large tumors that directly invade the transverse colon may present with colonic obstruction. Physical examination will reveal a palpable mass in up to 30% of patients.

Signs of Metastasis disease is found in 10% of patients.

The most common indications of distant metastasis are a palpable supraclavicular lymph node (Virchow node), a mass palpable on rectal examination (Blumer shelf), a palpable periumbilical mass (Sister Mary Joseph node), ascites, jaundice, or a liver mass. The most common site of hematogenous spread is the liver; tumor also frequently spreads directly to the lining of the peritoneal cavity

Gastric tumors may be associated with chronic blood loss, but massive upper gastrointestinal bleeding is rare. In Japan, the high incidence of gastric cancer has led to routine endoscopic screening; as a result, in that country, more than 50% of gastric cancers are diagnosed at an early stage.

STAGING SYSTEM

The Pathologic staging system currently in use worldwide is the American joint committee on cancer TNM staging system with the addition of the term R status to denote residual disease remaining after resection

TNM STAGING

The American joint committee on cancer (AJCC) has designated staging by TNM

classification.

PRIMARY TUMOUR

- **Tx :** Primary tumour cannot be assessed
- **T0**: no evidence of primary tumour
- Tis: carcinoma in situ: intra epithelial tumour without invasion of the lamina propria
- T1:Tumour invades lamina propria or submucosa
- T2:Tumour invades the muscularis propria or the submucosa
    1. T2a:Tumour invades muscularis propria
    2. T2b:Tumour invades subserosa
- T3: Tumour penetrates the serosa(visceral peritoneum) without invading adjacent structures
- T4: Tumour invades adjacent structures.

REGIONAL LYMPH NODES

- Nx: Regional lymph nodes cannot be assessed
- N0: No regional lymph node metastasis
- N1:Metastasis in 1-6 regional lymph nodes
- N2:Metastasis in 7-15 regional lymph nodes
- N3:Metastasis in more than 15 regional lymph nodes.

DISTANT METASTASIS

- Mx:Distant metastasis cannot be assessed
- M0:No distant metastasis
- M1:Distant metastasis.

AJCC STAGING

Stage 0 (Tis,N0,M0)

Stage 1a (T1, NO,MO)

Stage1b (T1,NI,MO;T2A,N0,M0;T2B,NO,MO)

StageI I (T 1,N2,MO;T2A,NI,MO;T2B,N1,M;T3,NO,MO)

StageIIIA (T2A,N2,MO;T2B,N2,MO;T3,NI,MO;T4,NO,MO)

StageIIIB (T3,N2,MO)

StageIV (T4,N1,MO; T4,N2,M0; T4,N3,MO; T1,N3,MO;T2,N3,M0; T3,N3,M0; Any T,ANY N,M1)

RESIDUAL DISEASE

R status described by Hermanek and wittekind in 1994 is commonly used to describe the tumour status in a patient following resection and is designated following pathologic evaluation of resection margins.

R_0 - No residual gross disease and Negative microscopic margins.

R_1 - Microscopic residual disease only

R_2 - Gross residual disease.

INVESTIGATIONS

UPPER GI ENDOSCOPY:

Differentiation between benign & malignant gastric ulcers at endoscopy can be difficult and several biopsies are taken (ideally six) from all parts of ulcer. Diagnostic accuracy approaches 100% if 10 samples are taken.

BARIUM MEAL

It gives a better impression of anatomy of tumour, and the degree of obstruction. It is also helpful for diagnosis of linitis plastica which may be missed at gastroscopy.

CT SCAN

Abdominal and pelvic CT is performed in the overall staging of patients. CT chest may be required for complete staging of proximal gastric tumours. The major limitations of CT as a staging tool are in the evaluation of early gastric tumors and small metastases in the Liver. The overall accuracy in determining tumors stage is 66% to 77%. CT can be used to determine nodal stage in 25% to 86% of patients.

ENDO ULTRASOUND

The overall staging accuracy of EUS is 75%. It correctly identifies T2 leisons (38.5%) of the time. It is better at identifying T1(80%) and T3(90%) lesions. The accuracy of EUS in the nodal evaluation is 65%.

LAPAROSCOPY

The Clinical benefit of laparoscopic staging after CT staging is that it identifies CT occult Metastatic diseases in 23% to 37% of patients.

PERITONEAL CYTOLOGY

Cytologic analysis of peritoneal fluid obtained by peritoneal lavage may identify occult carcinomatosis. Patients with positive peritoneal cytology findings have a prognosis similar to that of patients with macroscopic visceral (or) peritoneal disease(3 – 9 month median survival)

LYMPHATIC MAPPING:

Gives the essential role of nodal status in gastric cancer staging. Mapping agents such as radiocolloid with (or) without vital dye and activated carbon particles have been used. The identification rate varies from 90% from 100%.

POSITRON EMISSION TOMOGRAPHY:

Positron emission tomography, estimate tumour metabolism on the basis of the uptake of radiotracer – flurodeoxyglucose – used a staging tool for gastic cancer. This technique may reveal CT-occult metastases and may be used to assess response to Neoadjuvant therapy.

TUMOUR MARKER

CA 72-4 is highly specific to gastric cancer and may be more reliable as a tumour marker than CEA for gastric cancer. Elevated level of CA 72.4 is more frequent than CEA in stage III, IV patient and with Peritoneal metastasis.

H. PYLORI TESTS.

Tests	Sensitivity	Specificity
Histology (Geimsa stain)	93-99%	95-99%
Culture	72-92%	100%
Urease test	89-98% ;	93-98%
Urea breath test	95-98%	95 -98%
Serology ELISA	88-89%	86-95%
PCR	95%	95%
Stool antigen test	95-99%	90-95

SURGICAL TREATMENT

EARLY GASTRIC CANCER

Endoscopic Submucosal Resection

For mucosal cancers, approximately 3% will be expected to have positive lymph nodes and selectively justify the use of an endoscopic submucosal resection (ESMR). Patients with submucosal cancer, where the risk of nodal positivity can reach 20%, would be better considered for limited laparoscopic resections or limited open operations. Lymph node metastasis in both these situations is associated with larger tumor size, undifferentiated histology, and the presence of histologic lymphatic or blood vessel invasion. As a working rule lymph node metastases are rare if the tumor is less than 2 cm in diameter and well differentiated regardless of the depth of mucosal invasion.

Laparoscopic Resection

Laparoscopic resection is being used increasingly for early-stage cancer. This is

performed essentially by an extragastric approach after appropriate tattooing of the lesion endoscopically so as to ensure the ability to identify the lesion and the adequacy of the resection. The procedure can be prolonged, and it is essential that clear margins be obtained. Progressively more advanced procedures such as distal gastrectomy are being performed with the laparoscopic hand-assisted approach using a minilaparotomy

Proximal tumours

This tumours accounts for about 50% of all gastric carcinomas. These tumours are usually advanced at presentation and associated with poorer long term prognosis.

Siewert classification of OG junction tumours

Type I **:** Associated with Barrett oesophagus (or) true esophaged cancer growing into the OG junction

Type II **:** True junctional tumours that lie within 2cm of squamo columnar junction.

Type III **:** Cancer within subcardial region of stomach.

Type I **:** Treatment option for this tumour is oesophageal resection with 10 cm margin clearance.

Type II **:** oesophago gastrectomy

Type III **:** Total gastrectomy with reconstruction

There is no evidence that an extended gastric resection above and beyond complete clearance of the primary tumor improves survival; i.e., total gastrectomy does not improve survival over distal or proximal gastrectomy, provided that the tumor is removed completely with negative transection margins (R_0 resection). The extent of the gastrectomy therefore is site-

dependent and focuses on complete removal of the gastric carcinoma with preferably a 4- to 5-cm margin from the gross edge of the tumor. Clearly, anatomic limitations influence this margin because in antral lesions close to or involving the pylorus, only a limited portion of the duodenum can be removed. In similar fashion, a lesser extent of uninvolved esophageal margin may be acceptable provided complete histologic resection is possible.

Midbody Tumours

These account for 15% to 30% of all gastric cancers.

Treatement option for this tumours also total gastrectomy with regional lymphadenectomy.

Distal Tumour

These tumours account for approximately 35% of all gastric cancer. The standared operation for these lesions is distal subtotal gastrectomy with appropriate lymphadenectomy. Subtotal gastrectomy entails resection of approximately three fourths of the stomach, including the majority of lesser cuvature.

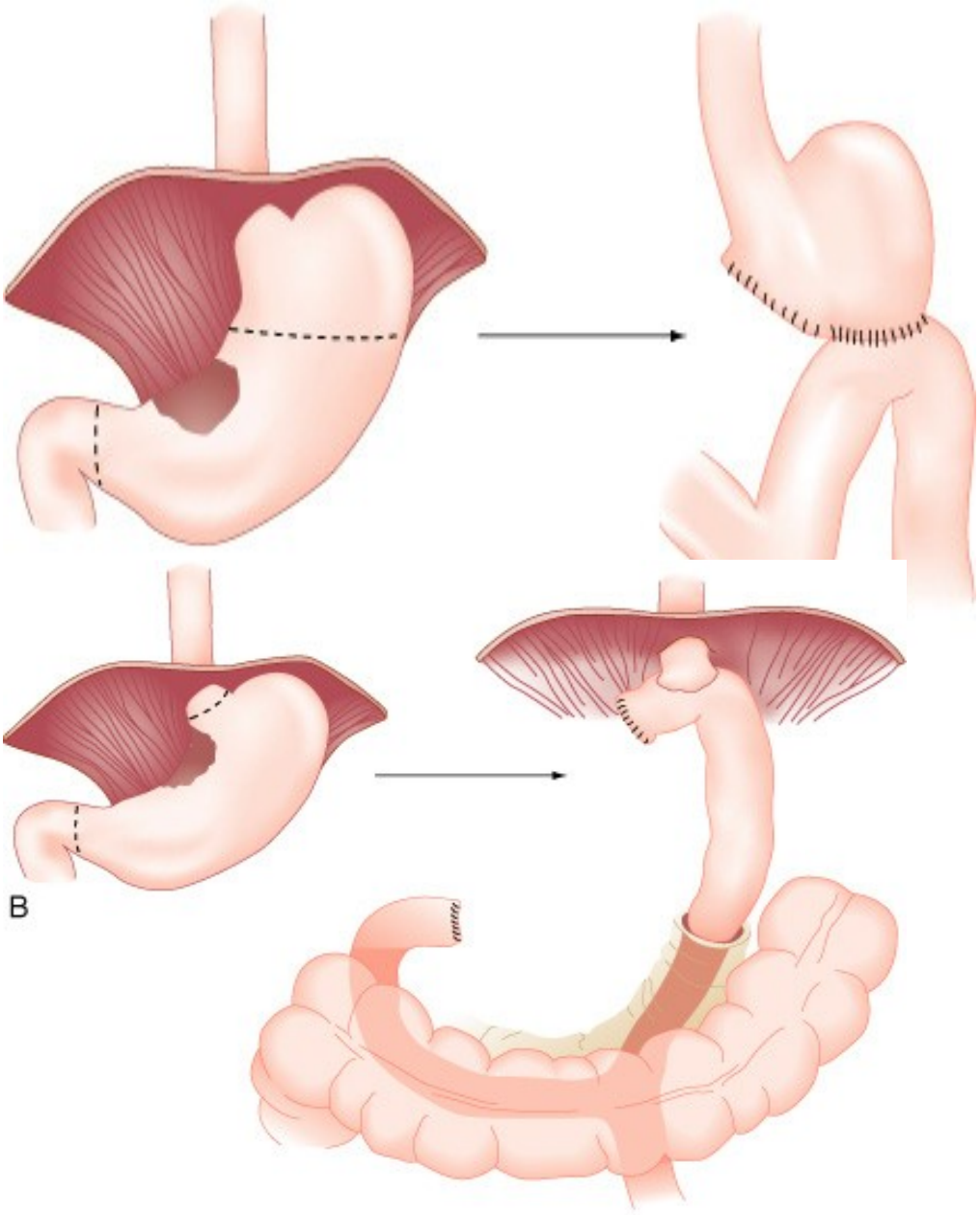
Extended organ resection is reserved for patients with apparently node-negative T4 lesions, in which complete resection requires resection of the invaded portions of the diaphragm, pancreas, spleen, adrenal gland.

SURGERY

TOTAL GASTRECTOMY:

(If the tumour involves the stomach diffusely or arises in the body of the stomach and extends to within 6 cm of the cardia or distal antrum) Removal of stomach with greater & leeser omentum along with nodes en bloc with Roux en y reconstruction. In a total gastrectomy, early mobilization of the left lateral segment of the liver will be necessary to evaluate the extent of a more proximal lesion .However, once the right side of the esophagus is isolated, then theesophagus can be encircled, and the left paracardial nodes and the paraesophageal nodes can be reflected inferiorly with the specimen In this situation, the short gastric vessels need to be divided. The left crus is skeletonized, and the left adrenal gland is identified commonly ,transect the stomach with the placement of a proximal Satinsky vascular clamp, which allows ready stabilization of the esophagus In proximal lesions, a frozen section of the esophageal margin is performed. In locally invasive extensive proximal lesions, the mucosal margin can be benign, but extraesophageal extension can be found in the periserosal tissues. Once this specimen is removed, the jejunal loop is isolated and divided with the GIA stapler .The small intestine for the Roux-en-Y anastomosis is divided with the stapler. Attention should be paid to ensure adequate vascularity of the limb, and some require careful transillumination to be sure that the arterial arcade is maintained without subsequent tension. The reconstruction can be performed by a direct end of

SUB TOTAL GASTRECTOMY WITH BILLROTH – II - ANASTAMOSIS



ROUX EN Y OESOPHAGO JEJUNOSTOMY

esophagus to jejunum anastomosis It is helpful in the sutured anastomosis to place a single stabilizing suture in the posterior aspect of the esophagus, suturing the posterior aspect to the

posterior surface of the jejunum, which stabilizes and aligns the appropriate. An alternative reconstruction uses the EEA stapler .

PROXIMAL GASTRECTOMY

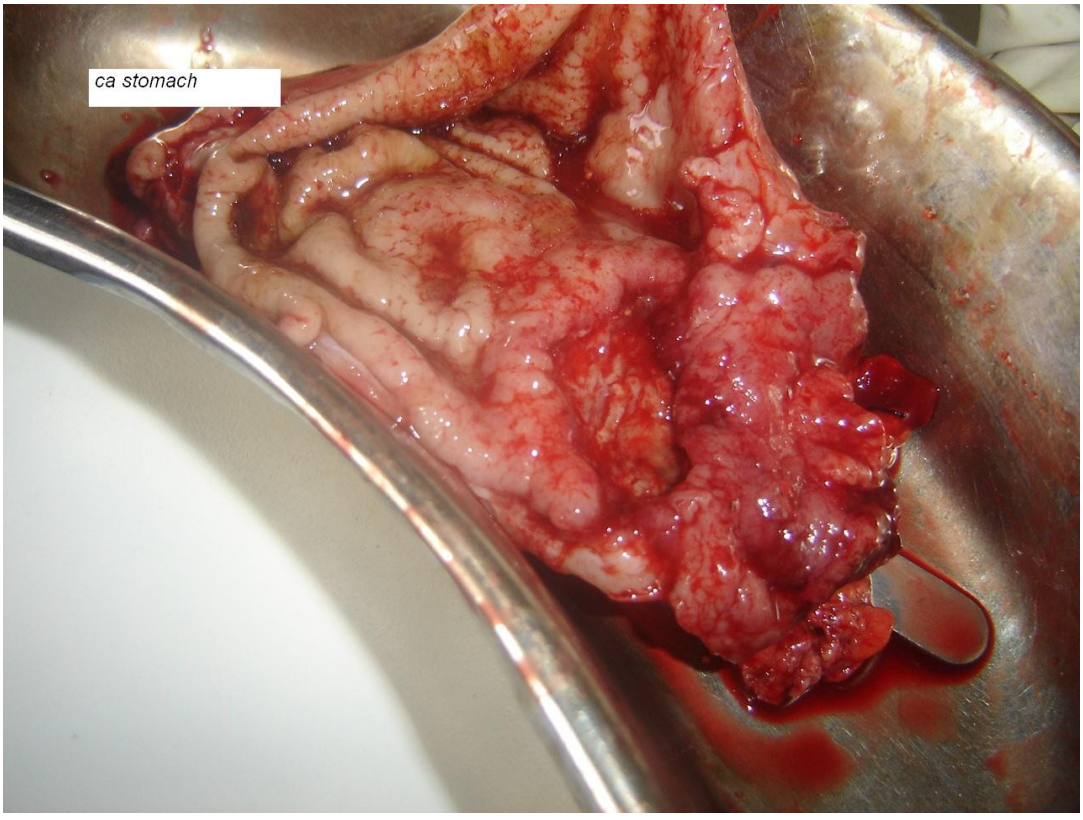
An simple midline incision is made in the abdomen

The left lateral segment of the liver is taken down and retracted, the esophageal hiatus is identified, and the soft tissue around the diaphragmatic hiatus is incised. The extent of the lesion is appreciated rapidly, and on occasion, local invasion into the crura of the diaphragm may need crural resection. The retroperitoneal attachments to the diaphragm on the left side then are incised, coming down toward the anterior surface of the left adrenal gland. Once the esophagus is mobilized, it can be retracted upward and forward, and a decision can be made as to the extent of resection required.

For cardiac lesions that are locally extensive, the spleen is mobilized and taken with the specimen. In more localized lesions, the short gastric vessels are taken close to the splenic hilum to enable greater mobilization. On the right side, the right crus is cleared, and all tissue is taken back to the celiac axis origin. The section then is carried forward along the anterior surface of the celiac axis to identify the left gastric artery. In the majority of

PARTIAL GASTRECTOMY

CUT SPECIMEN OF CA STOMACH



patients, all lymph nodes along the lesser curvature can be dissected readily and reflected back toward the proximal gastrectomy with the specimen. Care must be taken not to devascularize the lesser curvature. An appropriate distal site on the greater curvature is identified, and the stomach is divided with two passages of the GIA-60 or TA stapler. This can be done in an oblique fashion so as to create a relative tube of the distal stomach .

SUB TOTAL GASTRECTOMY

(If the proximal luminal resection margin is 5- 6 cm) .Removal of 80% of distal stomach leaving the short gastric vessels intact to ensure splenic preservation.Reconstruction is by either roux en y or billroth II or polya.

DISTAL GASTRECTOMY:

(Antral growth with 5cm marginal clearance) .Removal of less than 80% of stomach with reconstruction.

PALLIATIVE PROCEDURES

(For advanced gastric cancer to minimize bleeding and gastric out let obstruction)

- Gastrectomy
- Anterior gastro jejunostomy
- Laser or argon beam tumour ablation with laser recanalization.
- Endoscopic placement of coated metallic stents.

COMPLICATIONS OF SUGERY:

The most devastating complication is anastomatic leak, which occurs in 3% to 21% of

patients. Dumping syndrome will develop in 10% of patients.early dumping typically occurs 15 to30 minutes after meal and includes diaphoresis,abdominal cramps, palpitations and watery diarrohea. Late dumping is usually associated with hypoglycemia and hyperinsulinemia. The management include dietery habits and if refractory, a somatostatin analog.

Other complications are

- Bleeding – 0.3-5%
- Pulmonary 3-55%
- Infection 3-22%
- Anastomotic leak 3-21%
- Cardiac 1-10%
- Renal 1-8%
- Pul. Embolus -1-4%

OUT COME OF SURGERY

The overall 5 yr survival rate is 10-21 percent who undergo potentially curative resection has a slightly better prognosis with a 5 year survival rate of 24% to 57%. The 5 year survival rate who under go curative resection is 50% .The overall locoregional recurrence rate was 38% the median time to death from the time of recurrence was 6 months.

Neoadjuvant therapy

The advantages of Neoadjuvant chemotherapy is decreased tumour seedling at surgery and tumour sensitivity to a chemotherapeutic regimen.drugs used are etoposide, cis platin, 5-fluro uracil . The clinical response rate of combination of etoposide ,cisplatin and 5Fu range

from 21% - 31%.

Adjuvant chemo therapy

The adjuvant chemotherapy of 5Fu was associated with a survival benefit of 95%.

Post op External beam Radiation therapy

The combination of 5Fu with 40gy external beam radiation therapy versus RT alone showed improved survival. The three year survival rate for chemo RT were 52% compared to surgery only group 40%

Management of Advanced Disease

About 20-30% patients present with stage IV disease. The 5yr survival rate for patients with stage IV disease is zero. Because of this low cure rate and advanced disease at presentation, palliation is an essential component of gastric cancer Management.

Palliative surgery

Surgery achieves good palliation less than 50%of the tome. The procedure include palliative Distal gastrectomy with acceptable mortality rates. Palliative total gastrectomy & esophago gastrectomy should be approached with greater caution because the morbidity from these procedures is higher 4%.

Palliative chemotherapy

The Combination regimen included FAMTX(5Fu, doxorubicin, Methotrexate) FEMTX (5Fu, epirubucin, Methotrexate) and ELF(etoposide, leucovorin, &5Fu) patients who received combination chemotherapy had better survival.

Pallative Radiation therapy:

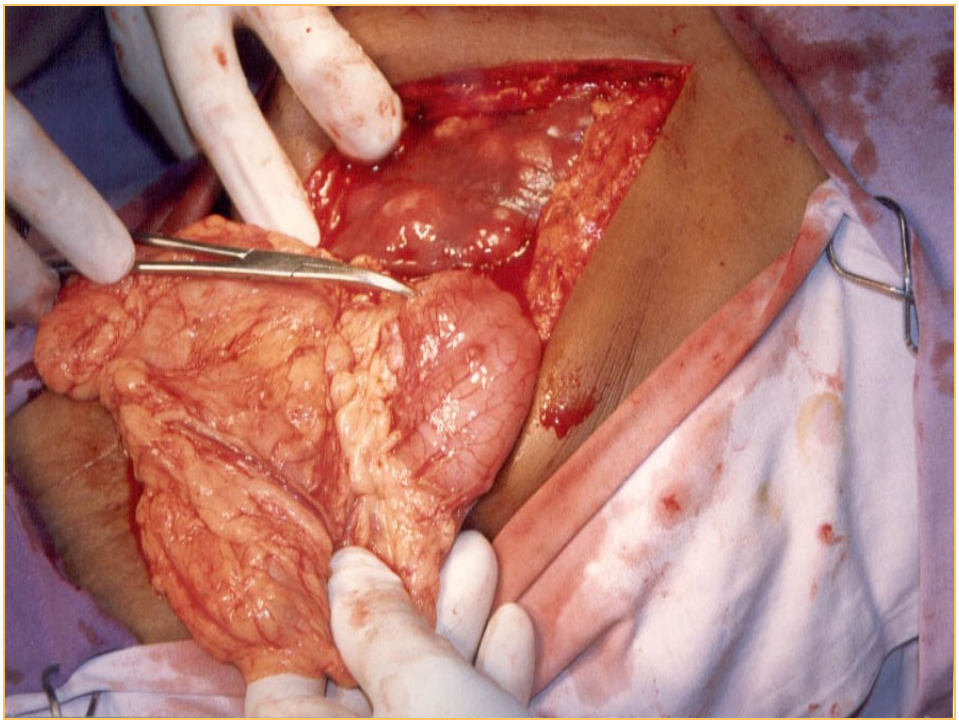
No large prospective trial has demonstrated a long term benefit from radiation therapy.

Palliation by Endoscopic means include laser recanalisation (or) simple dilation with (or) without stent placement can be used to treat obstruction.

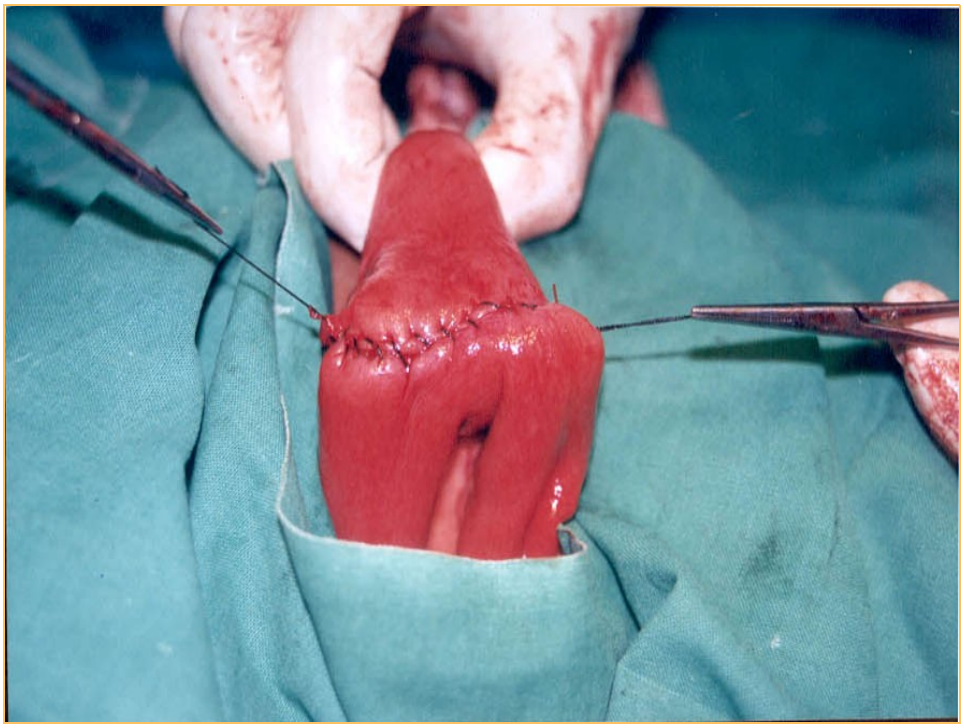
Intraperitoneal Hyperthermic Perfusion

This procedure is a combination of hyperthermia & Mitomycin C. Fujimoto et al reviewed that patients treated with perfusion survived longer(1 year survival rate 80% vs 34%).

CA STOMACH WITH LIVER SECONDARIES



OESOPHAGOJEJUNOSTOMY



Immunotherapy

Machara et al reported a combination of chemotherapy and intraperitoneal injections of OK-432 had a long survival rate. Other immunotherapy's are mycobacterium derived polysaccharides.

Gastric Lymphoma

The Stomach is the most common site of lymphoma in GIT. The average age of patients is 60 years.the most frequent site ofpresentation are pain, weight loss,bleeding and fatigue. H.Pylori appears to be a causative agent is the development of gastric lymphoma and MALT

lymphoma. Endoscopy detect the lesion is 80% of cases as sub mucosal infiltration.Other Lab test include lactate dehydrogenases and B2 Micro globulin determination. The pathological type of NHC is B cell Type & diffuse histiocytic type. Treatment include Acid Suppression therapy, Metrogyl and Antibiotic.Anti H. PYLORI treatment is necessary for eradication of H. p ylori. External beam irradiation and chemo therapy is offered to those who do not respond to an antiobiotic eradication and high risk patients. Gastric resection is rarely performed for pt. with MALT lymphomas.surgery is necessary in some cases to confirm diagnosis.Surgical resection is curative in patients with localized disease, although half of resected patients require chemotherapy.If surgery is performed,an attempt is made to resect the area grossly involved with lymphoma but leave grossly normal stomach intact. Negative margins are not necessary for cure. Surgery alone produces survival rate of 82%, where as radiation therapy produces five year survival rate of only 50%. 10- year survival rates of 80% have been seen in patients with Ann Arbor staging of stage IE and Stage II .Patient with lymphoma are initially treated with chemo therapy of CHOPP regimen. Radiation and surgery are reserved for patients who do not have a complete response to chemotherapy.

Gastric Carcinoid

Gastric carcinoid constitute only 2% of carcinoid tumors. gastric carcinoids are classified into three types . Type I carcinoids are associated with type A chronic atrophic gastritis, Type II carcinoids are associated with Zollinger-Ellison syndromewith multiple endocrine neoplasia syndrome-I, and type III are sporadic gastric carcinoids. Type I carcinoids are the most common type of gastric carcinoid and are found predominantly in

women. These patients have typically have elevated plasma gastrin levels and low gastric acid production. Type II gastric carcinoids are the least common type, accounting for 8% of cases with an equal distribution. Even they are associated with Zollingar Ellison syndrome in conjunction with MEN- I they share features with type -1 carcinoids,such as location in the fundus, multricentricity, elevated plasma gastrin level and small size.Type III carcinoids , which are sporadic carcinoids, represent 23% of cases and are found predominantly in men; The mean age of patients at diagnosis is 49 years. Patient may complain of histamine producing symptoms, such as cutaneous flushing,bronchospasm,itching and lacrimation. The sporadic tumours are single, solitary ,large and located in the antrum or fundus of stomach. The natural course of type III carcinoids is more aggressive with hepatic metastasis found at diagnosis in upto 50% of cases.

Gastric carcinoids are diagnosed by both biochemical and histologic means. Upper gastrointestinal endoscopy, including endo ultrasound, is also essential to evaluate the number, size ,extent and location of lesions. Extensive gastric biopsy should be performed in those with suspected gastric carcinoids, checking for histologic chromogranin, features of dysplasia,mucosal atrophy,and the degree of mucosal invasion.it appears that the rate of diagnosis correlates with the number of biopsys performed.CT of the abdomen is essential to exclude metastasis to the liver and nodal involvement.

The type of gastric carcinoid dictates the nature of treatment .for patients with either type I or type II carcinoids and with tumours less than 1 cm or with fewer than three to five lesions, treatment typically consists of an endoscopic polypectomy or endoscopic mucosal

resction.Antrectomy or local excision may be performed in a young patient with an elevated gastrin level who has recurrent or multifocal disease.. This treatment will decrese gastrin levels and frequently leads to the regression of the tumours. Older patients with many lesions may be followed if the lesions are small. For diffuse or recurrent disease, a completion gastrectomy may be recommended, depending on the patients age and course of the disease.Type III disease should be considered for en bloc resection with lympadenectomy, depending on the tumour size.It appears that five year survival rate for patients with type I and type II gastric carcinoids are between 60% and 75%.in contrast, the five year survival in patiets with type III gastric carcinoids is less than 50%. Follow up in patients with gastric carcinoids primarily consists of plasma chromogranin estimation. Plasma gastrin and urinary 5-HIAA levels may also be evaluated, although urinary 5-HIAA levels.

GASTRO INTESTINAL STROMAL TUMOURS

GISTs are the most common mesenchymal tumors of the stomach, with the stomach being the most common location of these tumors in the gastrointestinal tract (40%), followed by the small intestine (32%). GISTs of the stomach are predominantly found in males, with a median age at diagnosis of 63 years. The presenting features in these patients are varied and include bleeding (38%), abdominal pain (11.8%), and rupture (1%); 12% of patients have no symptoms. The median size of GISTs of the stomach is 6 cm.

Regardless of the location of these tumors in the stomach, R0 resection remains the treatment of choice, conferring a 5-year survival of 55%. Several studies have shown that the features recommended in the NIH guidelines, tumor size (>5 cm) and mitotic index (>5/50 hpf), are associated with a less favorable outcome. Tumor location in the gastroesophageal junction or fundus, ulceration, coagulative necrosis, and mucosal invasion were found to be associated with a poor outcome .Antral tumors, however, were found to be associated with a more favorable outcome .Those patients with unresectable or metastatic disease are offered treatment with Imatinib (Gleevec, an oral tyrosine kinase inhibitor). They may then be re-evaluated for potential resection if they demonstrate response to treatment.

OBSERVATION

- Cancer stomach is one of the commonest abdominal malignancy in our region.
- The incidence of distal growth continues to be increasing in our region.
- The incidence of CA stomach in distal part usually present with age above 50yrs.
- Cigarrete smoking showed increase in proximal cancers when compared distal cancers.
- The association between Blood group A and Carcinoma Stomach correlate with the study of AIRD ET AL.
- Endoscopy and Biopsy have a high sensitivity in picking up all subsite growths.
- The intestinal type and in association with distal growth have a favourable prognosis in operable cases.
- Stage 4 disease is common in proximal cancers which shows poorer prognosis in over all survival
- Gastrectomy with its acceptable morbidity is associated with good quality of life, whenever the tumour is operable
- Total Gastrectomy carries a high morbidity and mortality when compared to subtotal and partial Gastrectomy

CONCLUSION

- The causes of Distal stomach cancer continues to be commoner than proximal cancers among Indians.

- The Etiology of carcinoma stomach tends to be different etiology in both proximal and distal cancers.

- H.Pylori has a 45 .5% association of ca stomach and it is mostly associated with distal growths.

- Endoscopy has a high sensitivity in detecting all subsite growths but least sensitivity in detecting early cancers.

- Most cases present with stage 4 disease which shows absence of screening of this disease.

- Curative subtotal and distal gastrectomy is associated with good quality of life whenever the tumour is operable.

- Mortality is due to increase in duration of surgery and associated co-marbid illness and anastomotic leak.

S.	Name	Rel	IPNo.	Age/	SE	SM	AL	Diet	Subsite	Pain	Vom/	Mass	L/N	L/A

MASTER CHART

No				Sex							DYSP HAGIA			
1	Asokan	H	17640	65/M	800	√	√	M	Antrum	√	√			√
2	Pattanaasari	H	47730	60/M	800			M	Antrum	√	√		√	
3	Gurusamy	H	30628	50/M	800			M	Antrum	√	√			
4	Rajammal	H	28355	50/F	800			M	Antrum	√	√			√
5	Kalliammal	H	33605	55/F	800			M	Antrum	√	√	√		√
6	Pillaikani	H	21600	54/M	800	√		M	Antrum					
7	Mayillappan	H	14828	64/M	800	√		M	Antrum		√	√		√
8	Ayyadurai	H	2733	45/M	800	√	√	M	Antrum	√	√			√
9	Palaniappan	H	281395	62/M	800	√	√	M	Antrum	√	√		√	√
10	Muppidathi	H	35131	55/M	800			M	Antrum	√	√			
11	Muhtukutti	H	19729	49/M	800			M	Body(LC)		√	√	√	√
12	Murugan	H	26428	55/M	800	√	√	M	Antrum	√	√	√	√	√
13	Babu	H	36204	67/M	800	√		M	Antrum	√	√			√
14	Muthiahdevar	H	35208	65/M	800	√		M	Body(GC)			√		
15	Thiraviampillai	H	5705	70/M	800			M	Antrum	√	√	√		√
16	Ravikumar	H	37841	24/M	800			M	Antrum	√	√			
17	Mohammed	m	36686	47/m	800	√	√	fat	antrum	√				√

S. No	dehydration	MassPalpable	FullnessVague	VGP	Liver	GB	Ascites	Pelvic Deposits	BL GP&TY	USG/CT	Endoscopy	Biopsy	Bameal	H PYLORI	
1.	√								A+ve	Antral wall thickening	Antral Growth Goo	Adeno ca			Antral LN+ Fixed
2.				√					AB+ve	Antral wall thickening	Antral Growth Goo	Adeno ca			Antral G 2° A
3.				√					O+ve	(N)	Antral Growth	Adenoca			Antral Mes dep
4.		√ (E)		√					B+Ve	(N)	(N)	-			Growt
5.		√ (RHC)		√					AB+ve	Antralwall thickening 2° Liver Ascites	Antral Growth	Adeno ca			Grow 2° A
6.	√	√ (E)		√					AB+ve	Antralwall thickening	Antral Growth	Adeno ca			Antral L
7.	√			√					B +ve	Antralwall thickening para ao rtic LN	Antral Growth				Antral LN fixed t TC, PA,
8.	√								B+ve	Antral wall thickening	Antral growth	Adeno carcinoma			Antral LN infi par
9.	√			√					B+ve	Dilated stomach	Pre pyloric ulcer	Adeno carcinoma			Antral Peri
10.				√					B+ve	Duodenal thickening	Antral Growth & Goo	Adeno carcinoma			Antral C
11				√					B +ve	Antral wall thickening	GOO	Adeno carcinoma			Grou Pancreas
12		RHC		√	3cm				B +ve	Antral wall thickening, PALN,2°Liver	Antral Growth & Goo				Grou Pancreas TC,LN,
13		E		√					O+VE	Antral wall thickening	Antral Growth				Antral Mes dep
14		E					√		A +ve	Growth Body Ascites 2° liver	Growth Body	Adeno carcinoma		+	Grow Ascites
15		E		√					O +ve	Antral wall thickening,PALN,	Pyloric Antrum	Adeno carcinoma			Growth A to pancr Mes dep
16.	√	LHC							A +ve	N	Antral Growth	Poorly differentiated Adeno carcinoma			Growth A to pancr
17															

18	Natarajan	H	39830	58/M	800	√	√	M	CARDIA	√	Dysph agia			√
19	Samuvel	C	35767	50/M	800	√	√	M	Antrum	√	√			
20	Mariappan	H	14578	64/M	800	√	√	M	Antrum		√			
21	Muthiah	H	23988	57/M	800	√	√	M	Antrum	√	√			√
22	Shanmugavel	H	27093	61/M	800	√		M	Antrum	√	√	√		
23	Muthiahdevar	H	26818	61/M	800	√	√	M	Antrum	√				
24	Soundarrajan	H	27778	40/M	800			M	Antrum	√	√			
25	Balaiah	H	21981	55/M	800	√		M	Antrum	√	√			√
26	Vellammal	H	3988	55/F	800			M	Antrum	√	√	√		√
27	Velu	H	15037	68/M	800			M	Antrum	√	√		√	√
28	Ramakrishnan	H	51154	60/M	800			M	Antrum	√	√			√
29	Kanagaraj	H	18898	45/M	800	√	√	M	Antrum	√	√		√	√
30	Velkani	C	18974	52/F	800			M	Body(GC) LINITIS		√	√	√	√
31	Seenidurai	H	20148	50/M	800	√	√	M	Body LINITIS		√	√	√	√
32	Muthupandi	H	24804	65/M	800	√		M	Antrum	√	√			√
33	Lakshmi	H	30615	50/F	800			M	Antrum		√		√	√
34	Narayanan	H	21064	60/M	800			M	Antrum	√	√			√
35	Ganapathi	H	14009	50/M	800	√		M	Antrum	√	√			√
36	Alphonse	C	13893	50/M	800			M	Antrum	√	√			√

18	√			√					O+ve	Mass body of stomach	Body LC				Growth A to pancr Mes dep
19	√			√					O+ve	Ectopic Kidney GB Calculus	Growth Antrum				Growth A to panc
20				√					B +ve	Distended Stomach	Growth Antrum				Growt
21	√			√					O+ve	Antral wall thickening	Growth Antrum				Growth A to pancr
22	√	E					√		O +Ve	Antral Growth PALN	Growth Antrum	Poorly differentiated Adeno carcinoma			Growth A to pan
23		E		√					O+ve	Antral wall thickening 2°Liver	Growth Antrum	Adeno carcinoma			Gr Antrur LN,A
24				√					A+ve	N	Antral Growth				Growt mes LN,
25		E			4cm		√		O+ve	Antral wall thickening 2°Liver Ascites	Antral Growth GOO				Growth As
26	√	E		√					A+ve	Antral wall thickening	Antral Growth GOO			+	Growth A to
27	√		E	√					B+ve	Duodenal Pathology	Antral Growth				Growth A As fi
28			E	√					B+ve	Antral Wall thickening	Antral growth				Growt LN A
29		LHC					√		AB+ve	Antral growth LN ascites +	Anthral growth	Poorlydiffer Adeno carcinoma			Gr Antrur L
30	√	LHC					√		AB+ve	ascites	Anthral Growth, Linnitus, plastica	Poorlydiffer Adeno carcinoma	√		Contracte Stoma Ova
31	√	E(RHC)					√		A +ve	Mass epigastrium	Linitis Plastica	Adeno ca	√		Antra Body t
32	√	E			4 cm				B+ve	Antral Wall thickening	Antral Growth	Mucinous Adeno ca			Antral Py
33		E							AB+ve	Antral Wall Thickening,2° liver	Antral Growth				2° Antral Gr
34	√		E	√					AB+ve	Antral Wall LN, Ascites +	Antral Growth				Antra to Bod pancre
35	√		E	√					A +ve	Ascites GOO MRD	Antral Growth	Adeno ca			Growt As
36				√					O+ve	Antral Wall Thickening	Antral Growth				Antra

37	Shamugathai	H	37868	58/F	800			M	Antrum	√	√	√		
38	Sudalai	H	39760	74/M	800	√	√	M	Antrum	√	√			√
39	Arunachalam	H	3095	60/M	800			M	Antrum	√	√	√		
40	Vedhamanikam	H	49220	55/M	800			M	Antrum	√	√			√
41	Thirumaki	H	16078	63/M	800	√	√	M	Antrum	√	√			
42	Ramalakshmi	H	22240	47/M	800			M	Antrum	√	√		√	√
43	Dharmar	H	36087	58/M	800	√	√	M	Antrum	√	√		√	√
44	Thirumalai	H	16078	63/M	800	√	√	M	Antrum	√	√		√	√
45	Ramaih	H	27873	40/M	800	√		M	Antrum	√	√			
46	Grace	C	18080	45/F	800			M	Antrum	√	√			
47	Andidevar	H	31637	60/M	800	√		M	Antrum	√	√	√		
48	Narasiman	H	9276	55/M	800	√		M	Antrum		√	√		√
49	Nagalingam	H	25936	54/M	800	√		M	Antrum		√	√	√	√
50	Rawathy	H	28113	50/F	800			M	Antrum				√	√
51	Ramachandran	H	52332	40/M	800			M	Antrum	√	√			√
52	Muthammal	H	33603	57/M	800			M	Antrum	√	√			√
53	Ayyanu	H	50078	65/M	800	√	√	M	Antrum	√	√	√		√
54	Msooth	M	31106	60/M	800	√		M	Body(GC)	√	√	√		
55	Subbiah	H	36270	75/M	800	√	√	M	Antrum	√				

37	√	LHC							O+ve	2° liver	Antral Growth	Mod. diff Adeno ca			Antral 2° liver par
38	√			√					O+ve	Antral Wall Thickening,Ascites	Antral Growth				Antral Fixed As
39		E		√					O+ve	Antral Wall Thickening	Antral Growth Goo	Adeno ca			Antral Fixed
40				√					O+ve	Antral Wall Thickening	Antral Growth	Adeno ca			Antral
41	√		E	√					B+ve	Antral Wall Thickening	Antral Growth				Antral Perige
42	√		E	√					B+ve	Antral Wall Thickening	Antral Growth				Antral C
43		E							O+ve	Antral Wall Thickening					Antral C
44		E		√					A+ve	Antral Wall Thickening	Antral Growth				Antral Gro mes
45				√					B+ve	N	Antral Growth	Adeno ca			Antral
46				√					B+ve	N	Antral Growth	Adeno ca			Antral C
47		ERHC		√					A+ve	Antral Thickening ascites +	Antral	Adeno ca			Antral C As
48		E			4 cm				AB+ve	Antral Thickening Liver Ascites	Antrum	Poorly Diff Adeno ca			Antral C Liver
49		E			3 cm				B+ve	Antral Thickening	Antrum				Antral C Liver L
50	√	E		√	3cm				AB+ve	Antral Thickening 2° Liver	Antrum	Adeno ca			Antral C 2°Live Par
51			√E						O+ve	Antral Thickening	Antral Growth				Antral Gr
52		√E					√		AB+ve	2° Liver ascites	Antrum Body				Antral asc panc
53				√			√		B+ve	Antral Thickening	Antral Growth				Antrur
54	√	E	E						AB+ve	Antrum Thickening 2°liver	N				Antrum E
55	√								O+ve	Ascites R+Pl Effu……..	N				Antrur

56	Muthah ayyar	H	31878	67/M	800	√		V	Antrum	√	√	√		
57	Durairaj	H	25389	56/M	800	√	√	M	Antrum	√		√		√
58	Muthiah	H	218324	84/M	800			M	Antrum	√	√	√	√	√
59	Sudalai	H	14342	64/M	800	√		M	Antrum	√	√	√		√
60	Sankaranaryan	H	38225	35/M	800	√	√	M	Antrum	√	√		√	√
61	Yesuvadian	C	3550	65/M	800	√	√	M	Antrum	√	√		√	√
62	Ramasubhu	H	20315	55/M	800	√	√	M	Antrum	√	√			
63	Subramanian	H	19426	70/M	800	√	√	M	Antrum	√		√		√
64	Muthusamy	H	91484	45/M	800	√	√	M	Body(Gc)	√				√
65	Murugan	H	20326	50/M	800	√	√	M	Cardia	√	Dysphagia	√	√	√
66	Narayanan	H	19252	45/M	800	√	√	M	ANTRUM	√	√			
67	Abraham	C	21382	54/M	800	√	√	M	AntrUM	√	√			√
68	Mahalingam	H	24529	38/M	800	√	√	M	Antrum	√	√			
69	Jeyankani	H	14206	48/F	800			M	Body(LC)	√	√	√		
70	Rangitham	H	22236	46/F	800			M	Body(GC)	√				√
71	Paramasivan	H	20313	60/M	800	√		M	Antrum	√	√			√
72	Ramasubbu	H	4298	43/M	800	√		M	OG jn	√	Dysphagia			
73	Ganapathy	H	40239	72/M	800			M	Body(GC)	√				√
74	Arumugam	H	30803	58/M	800	√		M	Antrum	√	√			√

56	√	E			2 cm				O+ve	Antrum Thickening 2˚ liver ascites	Antral Growth				Antrum liver p
57		E			2cm		√		O+ve	Antrum Thickening 2˚liver	Antral Growth				Antrum l
58	√	E			4cm		√		B+ve	Antral 2˚liver LN	Antral Growth				Antrum As
59		E			3cm				AB+ve	Antrum 2˚Liver LN	Antral Growth				Antrun LN A
60							√		B+ve	Antralwall thickening	Antral Growth				An
61	√		E				√		B+ve	Antralwall thickening	Antral Growth	Adeno ca			Antra LN+ Fixe
62			E				√		O+ve	Antralwall thickening	Antral Growth	Adeno ca			Antral G mesente
63	√	E	E				√		B+ve	Antralwall thickening ln +	Antral Growth				Antr As
64			LHC						AB+Ve	(N)	Growth Body GC	FNAC LN Adeno ca			Grow
65	√	E							A+ve	(N)	Fundal growth				Growt
66	√	E					√		B+ve	Antral growth	Antral Growth	Adeno ca			Antrum
67	√			√					O+ve	Antral thickening	Antral Growth	Adeno ca			Antral L
68			√						AB+ve	Normal	Body LC	Adeno ca			B
69			E						A +ve	Growth	Body LC	Adeno ca			Bo
70									O+ve	Antral wall thickening	Body LC				Bo
71			E	√					A+ve	Antral wall thickening	Antral Growth	Adeno ca		_	Antrum
72		E							AB+ve	N	Cardiac Growth	Adeno ca			Ca
73			E						A+ve	Antral wall thickening	Body GC	Adeno ca		+	Body G
74		E							A+ve	Antral wall thickening	Antrum	Adeno ca			Antral Gr Pancreas

75	Murugan	H	25549	49/M	800	√		M	Body(GC)	√				
76	Lakshmi	H	99969	47/F	800			M	Antrum	√	√			√
77	Ganapathy	H	32797	62/M	800			M	Antrum	√	√			√
78	Subbiah	H	23465	65/M	800	√	√	M	Antrum	√	√	√		√
79	Shanmugam	C	26217	60/F	800			M	Linitis plastica	√	√			
80	Kandiahdevar	H	49520	60/M	800	√		M	Antrum	√	√			
81	Mani	H	17314	60/M	800	√		M	Antrum	√	√	√		
82	Ganapathy	H	31909	66/M	800	√		M	Antrum	√	√			√
83	Narasingamal	H	21870	30/F	800			M	Antrum	√	√			
84	Mani	H	17414	60/M	800	√	√	M	Antrum	√	√	√	√	√
85	Sappani	H	49413	43/M	800	√		M	Body(GC)	√				
86	Subramani	H	17575	55/M	800	√	√	M	Antrum		√		√	√
87	Mariammal	H	39539	67/F	800			M	Body(GC)	√	√		√	√
88	Ganapathy	H	60604	67/M	800	√	√	M	Antrum	√	√	√		√
89	Manivelraj	H	5434	40/M	800	√	√	M	Antrum	√	√	√	√	√
90	Valliammal	H	57286	65/F	800	√	√	M	OG JN	√	Dysphagia			√
91	Thriumalai	H	34679	67/M	800	√	√	M	Antrum		√			√
92	Arjunan	H	37623	50/M	800	√		M	Antrum	√	√	√		
93	Thayammal	H	33860	55/F	800			M	CARDIA		Dysphagia	√		

75			E						O+ve	Antral wall thickening	Body GC	Adeno ca			B
76			√						B+ve	Antral wall thickening	Antrum				An
77		E							O+Ve	Antral wall thickening	Antrum	Poorly differentiated Adeno ca			An
78		E			2 cm				A+ve	Antral wall thickening, 2˚ Liver PALN	Antral Growth				Antrum
79		E		√					O+Ve	Antral wall thickening, 2˚ Liver		Adeno ca			2˚ Live
80		E		√					A+ve	Antral wall thickening, 2˚ Liver	Antral Growth	Adeno ca			Antrum 2
81		√					√		O+ve	Antral Thickening ascites	Antral Growth				Antrum
82	√		E						B+ve	Antral wall thickening	Antral Growth	Adeno ca Well differentiated			Antr
83				√					B+ve	Antral wall thickening	Antral Growth				Antr
84	√	E		√	4cm		√		A+ve	Antral wall thickening	Antral Growth				Antrum
85			E						O+ve	N	CT Body GC				Body
86		E					√		A+ve	N	GOO	Adeno ca Welldifferentiated			Antrum PE Rou
87	√			√					O+ve	Antral wall thickening	Antral Growth	Adeno ca			B
88	√	E			2cm				O+ve	Antral wall thickening,2˚ liver	Antral Growth				
89		E			3cm		√		B+ve	Antral wall thickening	Antral Growth				
90		E		√			√		AB+ve	N	Body GC				Growth B +PD +P pan
91	√	√		√			√		O+ve	Free Fluid LN Antral Growth	Antral Growth	Adeno ca			Liver 2° A TC in
92	√	√		√			√		B+ve	Free Fluid LN Antral wall thickening	Antral Growth	Adeno ca			Growt Ascitic f De
93		√			3cm				B+ve	Hepatomegaly IHBR dilatation PH nodes	Growth Body of stomach	Adeno ca			Growt stomach no

94	Arumugam	H	26655	52/M	800	√	√	M	Antrum	√	√			√
95	Thangaraja	H	34538	60/M	800	√	√	M	Antrum	√	√			
96	Muthusamy	H	41489	45/M	800	√		M	Body(GC)	√				√
97	Petchikumar	H	49496	73/M	800	√	√	M	Antrum	√		√		
98	Venkatachalam	H	49485	52/M	800	√		M	OG JN		Dysphagia	√		
99	Ganesan	H	7903	64/M	800	√	√	M	ANTRUM	√				
100	Pandi	H	5161	45/M	800			M	Body(GC)	√				
101	Selvaraj	H	6158	57/M	800	√		M	Growth Cardia		Dysphagia			
102	Syedmohamed	M	31863	60/M	800	√		M	Pyloric growth	√	√	√		
103	Paramasivan	H	29576	65/M	800			M	Antrum	√	√	√		√
104	Joseph	C	36392	65/M	800	√		M	Antrum Growth	√	√	√		
105	Vairamuthu	H	62501	60/M	800	√	√	M	Antrum	√	√	√		
106	Ganesan	H	7903	64/M	800	√		M	Antrum		√	√		
107	Krishnan	H	39822	65/M	800	√	√	M	Antrum		√	√		√
108	Abdulrahim	M	40853	65/M	800	√	√	M	Antrum	√	√	√		
109	Manivelraj	C	51286	40M	800	√	√	M	Antrum	√	√	√		
108	Perumal	H	48604	35/m	800	√	√	m	antrum	√				√
111	Cristopher	C	48670	45/M	800	√		M	Body(LC)	√		√		√
112	Mohamed bashir	M	36636	47/M	800	√	√	M	Antrum	√	√			√

94	√			√					A+ve	Antral wall thickening	Antral Growth	Adeno ca		+	Growth
95	√	√		√					B+ve	Antral wall thickening	Antral Growth	Adeno ca			Growth
96									AB+ve	Normal study	Elevated mucosa irregularity				Growt
97	√	√		√	2cm		√		B+ve	Ascites hepaticadenopathy	Antral Growth	Adeno ca			2° Live Growt Pancreas
98	√	√			3cm		√		B+Ve	Liver 2°	Growth OG jn			√	Growth no
99	√	√		√					A+ve	Antral wall thickening	Antral Growth	Adeno ca			Antral Gr infil
100		√							O+ve	Liver 2° PH nodes	Growth Body of stomach	Adeno ca			Growth stomach
101	√		√						A+ve	N	Cardia growth	Signet ring type			Growt LN,Omen
102	√	√		√	√		√		B+ve	Antral wall thickening	Antral Growth	Adeno ca			Antral
103		√		√					B+ve	Antral wall thickening	Antral Growth	Adeno ca			Antral
104	√			√					AB+ve	Distended stomach	Antral Growth				Antral
105		√							A+ve	Antral wall thickening	Antral Growth	Adeno ca			Antral C
106		√					Dvt Lt LL		A+ve	Antral wall thickening	Antral Growth	Adeno ca			Antral Gr infil
107	√		√						B+ve	Distended stomach	Growth Body			_	Growth b
108	√			√					AB+ve	Antral wall thickening	Antral Growth	Adeno ca			Antral C
109	√			√					B+ve	Antral wall thickening	Antral Growth	Adeno ca			Antral C
110	√				√		√		O+ve	N	Body of Stomach				Diffuse PD+O
111	√	√	√						B+ve	N	Antral Growth	Spindle cell ca			Grow
112		√		√					AB+ve	Antral wall thickening	Antral Growth				Antral C As

113	Sermakani	H	3927	38/F	800			M	pyloric	√	√		√	√
114	Ramar	H	16634	48/M	800	√		M	Antrum	√	√	√		√
115	Anandha naryanan	H	25798	66/M	800	√	√	M	Diffuse	√			√	√
116	Thangaraj	H	28985	42/M	800	√	√	M	Antrum	√	√			
117	Maharaja	H	32200	35/M	800	√	√	M	Antrum		√			
118	Sermadevi	H	33087	35/M	800	√		M	Antrum	√	√	√		
119	Ramalingam	H	26012	65M	800	√	√	M	Antrum	√	√		√	√
120	Thayammal	H	33860	60/F	800			M	Antral growth	√		√		√
121	Krishnan	H	39822	65/M	800			M	Antrum	√	√	√		√
122	Abdulrahim	M	40853	65/M	800	√	√	M	Antrum	√	√			√
123	Ganthimathi	H	23160	70/F	800			M	Antrum		√			
124	Arumugam	H	26615	52/M	800	√		M	Antrum	√	√	√		√
125	Perumal	H	28760	52/M	800		√	M	Antrum	√	√			
126	Murugesan	H	31863	62/M	800	√	√	M	Antrum		√	√		√
127	Ponnusamy	H	34769	60/M	800	√		M	Antrum	√				
128	Arjunan	H	3762	57/M	800	√	√	M	Antrum		√			
129	Thangalakshmi	H	43811	57/F	800			M	Antrum	√	√			√
130	Sarumathy	H	45465	40/F	800			M	Antrum	√	√	√		
131	Murugan	H	15161	31/M	800	√		M	Antrum	√	√	√		
132	Krishnavel	H	18433	31/F	800			M	Antrum	√			√	√

113	√				√		√		B+ve	Antral wall thickening	Antral Growth				Antra
114	√	√		√					B+ve	Antral wall thickening	Antral Growth				Antra
115	√		√		√		√		O+ve	Ascites	Diffuse involvement stomach	P00rly diffadenoca	√		Contract LN A
116	√			√					B+ve	Antral wall thickening	Antral Growth				Antral G
117		√		√					A+ve	Antral wall thickening	Antral Growth			_	Antral G
118	√	√		√					B+ve	Antral wall thickening	Antral Growth				Antral G
119	√	√		√					O+ve	Antral growth					Antral G
120	√	√							AB+ve	Antral wall thickening	Body and Antral Growth				Growth
121	√	√	√						O+ve	Antral wall thickening	Antral Growth	Adeno ca			Antral G
122	√		√						B+ve	Antral wall thickening	Antral Growth	Adeno ca			Antral G As
123	√		√			√			AB+ve	Antral wall thickening	Antral Growth	Adeno ca			Antral G Liver
124		√	√						A+ve	Antral wall thickening	Antral Growth	Adeno ca		+	Antra
125	√	√		√		√	√		O+ve	Ascites 2° Liver	? Linitis plastica	Adeno ca	√		Diffuse
126	√	√							A+ve	Antral wall thickening	Antral Growth	Adeno ca			Antral G
127			√						AB+ve	Antral wall thickening	Antral Growth				Antral Gr
128			√						B+ve	Antral wall thickening	Growth Body	Adeno ca			Growth B
129									A+ve	Antral wall thickening	Antral Growth	Adeno ca		+	Antra
130									O+ve	N	Antral Growth				Serosa r
131	√	√		√					O+ve	Antral wall thickening	Antral Growth	Adeno CA			Antral G
132		√							B+ve	N	Antral Growth	Adeno CA			Antral G

133	Thangathai	H	29312	50/F	800			M	Body of Stomach	√				√
134	Nallamadan	H	51675	70/m	8oo			M	Antral growth	√	√			
135	Vellammal	H	30566	70/F	800			M	Body of Stomach	√	√	√		√
136	Thangaraja	H	34538	60/M	800	√	√	M	Growth Antrum	√	√			√
137	Selvaraj	H	50590	65/m	800	√	√	M	Pyloric growth	√				√
138	Avudaithai	H	34221	45/F	800			M	Growth Antrum		√			√
139	Nainarammal	H	8591	50/F	800			M	Growth Antrum	√	√			
140	Deivakirubai	C	25414	35/F	800			M	Growth Antrum	√	√	√		√
141	Marial	c	48067	50/f	800			M	antrum	√	√	√		
142	Lashmanan	H	35426	62/f	800	√	√	M (F)	cardia	√	Dysphagia			
143	Chandran	H	7148	41/F	800				CARDIA	√	Dysphagia			
144	Jamilal	M	26987	53/F	800			FATTY MEAL	Growth Og jn		Dysphagia		√	√
145	Parwathy	H	27625	45/m	800			M	CARDIA		Dysphagia			√
146	Malliga	H	38154	38/F	800			M	OG JN		Dysphagia			√
147	Shaloni	H	28157	55/M	800			wt-69kg	OG jn		Dysphagia			
148	Rajagopal	H	38788	55/F	800			M	DIFFUSE	√			√	√
149	Selvaraj	H	51535	65/M	800	√	√	M	ANTRUM	√	√			
150	Marammal	H	44086	65/F	800			M	PYLORIC		√			√

133	√	√	√				√		AB+ve	Ascites +	Growth Body	Adeno CA			Growth B
134	√				√		√		B+ve	N	Antral Growth				Growt
135	√			√					A+ve	N	Growth Body	Adeno CA			Growth
136	√		√				√		B+ve	Antral wall thickening	Antral Growth	Adeno CA			Growth A Post
137				√					O+ve	Antral wall thickening	Antral Growth				Antral G
138		√		√					B+ve	Antral wall thickening	Antral Growth				Antral
139				√					B+ve	Antral wall thickening	Antral Growth	Adeno CA			Antral
140	√	√							O+ve	Antral wall thickening Ascites	Antral Growth	Adeno CA			Antral Gro 2°
141									A+ve	Antral wall thickening	Antral Growth	Adeno CA			Antral Gro 2°
142									A+ve	N	Cardia Growth	Adeno CA			Cardia
143									AB+ve	Ascites	Cardia Growth	Adeno CA			Growth 2° A
144									B+ve	-	Growth OG JN	Adeno CA			Oesoph
145									O+ve	N	Cardia and Fundus				Growth Fu
146									O+ve	Liver 2°	OG JN	Adeno CA			Growt Ascites
147									B+ve	N	OG JN	Poorly Diff Adeno CA			Growt Ascites
148									O+ve	Ascites	Growth Body	Poorly Diff Adeno CA			Diffuse i
149									A+ve	Antral Growth	Antral Growth	Adeno CA			Growth A
150									A+ve	Antral Growth	Antral Growth	Poorly Diff Adeno CA			Growth A

CA STOMACH PROFORMA

Name :
Age :
Sex :
Ip no :
DOA :
DOS :
DOD :
Unit :

Complaints

Mass abdomen : duration
Size
Site

Pain abdomen : duration
location
nature- colicky / dullaching /others

Jaundice : duration
obstructive/hepatocellular

Vomiting : nature food/bile/blood/others

Fever

H/o dyspepsia

H/o ball roling movements

Abdominal distension

H/o pruritis

bowel habits – constipation/diahorrea/melena/steatorrhea

H/o drug intake

Past history

DM/HT/TB/BA/IHD

H/O previous surgery

H/o jaundice

H/o blood transfusion

Personal history

Diet; veg/non veg/mixed/ fatty meals/smoked food

smoker / alcoholic

Menstrual history

Treatment history

General examination

Nutrition

Hydration status

Anaemia.

Jaundice.

Pedal edema.

Lymph nodes.

Vitals-PR : BP : RR : TEMP :

SYSTEMIC EXAMINATION:

PER ABDOMEN :

INSPECTION :

Site :

Size :

Shape :

Umbilicus :

Veins/pulsation

Moves with respiration.

PALPATION:

Consistency

Warmth tederness

Guarding/rigidity

Organomegaly

PERCUSSION:

Liver dullness

Over the mass

Free fluid

Shifting dullness

Fluid thrill

AUSCULTATION

Bowel sounds

DRE

OTHER SYSTEMS - CVS/RS/CNS/LOCOMOTOR

CLINICAL DIAGNOSIS

INVESTIGATIONS

HB%

TC

DC

ESR

URINE - alb/sug /dep

BLOOD - sug/urea/creatinine

SR - electrolytes-na,k

LFT - sr.bilurubin

SGOT/AST

SGPT/ALT

Sr.proteins - alb/glo/total

X RAY CHEST PA VIEW

XRAY ABDOMEN - supine/ erect

Endoscopy - upper gi endoscopy

HPE Report

H.PYLORI

OPERATION NOTES

POST OP FOLLOW UP

Day of ryles tube removal

Day of suture removal

Complications

Period of follow up - RT/CT

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